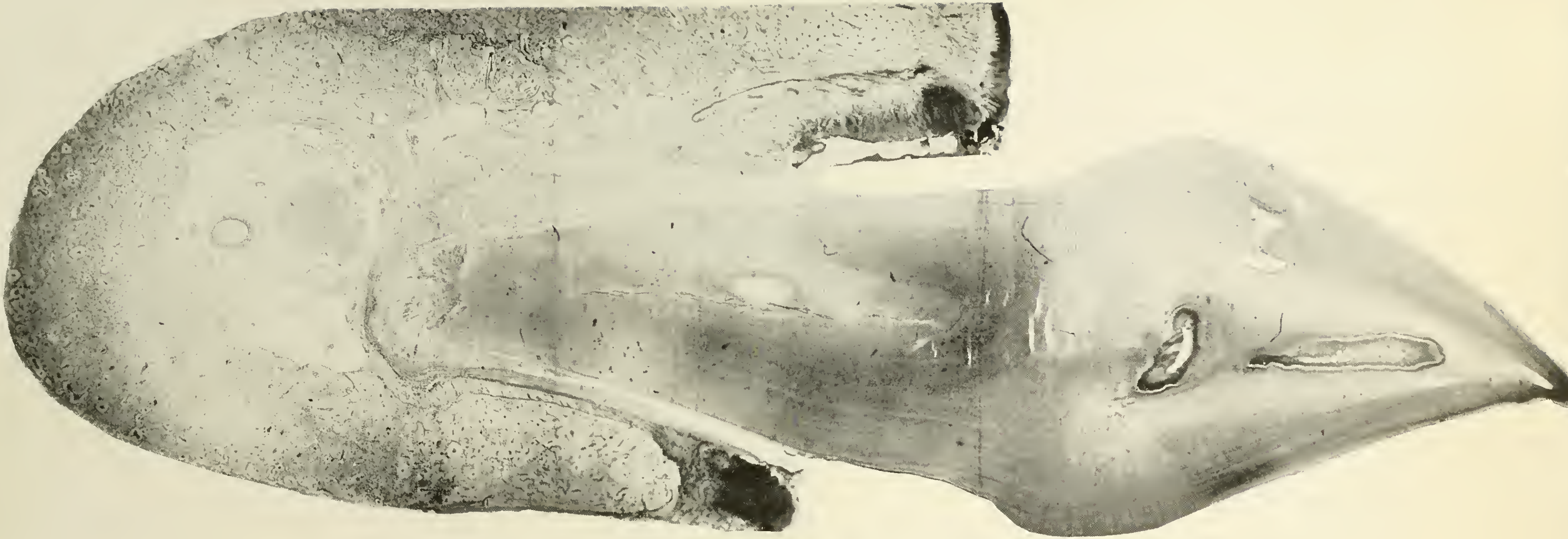


X 75. A. A. obj. Zeiss. In three sections. Micro-photograph reduced one-half size.

SECTION OF LOWER JAW OF TWELVE-YEAR-OLD DOG WITH CUSPID TOOTH IN SITU.

Nearly every phase of disease of tooth and jaw is here illustrated. The last stages of interstitial gingivitis with resultant pyorrhœa alveolaris appear, showing tooth attached by fibrous union for only a short distance upon one side near apex of root. Thickening of the cementum (cementosis), due to irritation and inflammation of the peridental membrance, is present. Cementoclasts (indistinctly seen) are producing absorption of the end of the root. Marked inflammation has occurred at gingival margin with pus pockets at the labial and lingual borders. Absorption of the lingual border appears directly upon the end. Absorption of the outer plate has occurred in a lateral direction for the entire length, from the root of the tooth outward, with obliteration of the pulp cavity.

INTERSTITIAL GINGIVITIS

AND

PYORRHŒA ALVEOLARIS

BY

EUGENE S. TALBOT, M. S., D. D. S., M. D., LL. D.

LATE PROFESSOR OF STOMATOLOGY, WOMAN'S MEDICAL SCHOOL, NORTHWESTERN UNIVERSITY
LATE LECTURER ON STOMATOLOGY, RUSH MEDICAL COLLEGE, UNIVERSITY OF CHICAGO; FELLOW
OF THE CHICAGO ACADEMY OF MEDICINE; FELLOWSHIP MEMBER OF THE NEW YORK STATE
DENTAL SOCIETY, 1908; SECRETARY OF SECTION ON STOMATOLOGY OF THE AMERICAN
MEDICAL ASSOCIATION; VICE-PRESIDENT AMERICAN MEDICAL ASSOCIATION, 1905;
MEMBER VII INTERNATIONAL MEDICAL CONGRESS, 1881, LONDON; HONORARY PRESI-
DENT X INTERNATIONAL MEDICAL CONGRESS, 1890, BERLIN; HONORARY PRESIDENT
XII INTERNATIONAL MEDICAL CONGRESS, 1897, MOSCOW; CORRESPONDING
MEMBER, BUDAPEST ROYAL SOCIETY OF PHYSICIANS; HONORARY PRESIDENT
INTERNATIONAL ASSOCIATION OF STOMATOLOGY, 1907, PARIS; MEMBER FIRST
FRENCH CONGRESS OF STOMATOLOGY, 1907, PARIS; HONORARY SECRETARY
PAN-AMERICAN MEDICAL CONGRESS, 1901, HAVANA; HONORARY MEMBER
ODONTOLOGISCHEN GESELLSCHAFT, BERLIN; HONORARY MEMBER
ASSOCIATION GENERALE DES DENTISTES DE FRANCE, PARIS; HONOR-
ARY MEMBER SOCIEDAD ODONTOLOGICAL ESPANOLA, MADRID;
CORRESPONDING MEMBER DANSK-TANDLAEGEFORENING, 1901;
HONORARY MEMBER STOMATOLOGY SOCIETY OF HUNGARY;
CORRESPONDING MEMBER OF THE ITALIAN STOMATOLOGICAL
FEDERATION, 1910; MEMBER CHICAGO ACADEMY OF
SCIENCES; MEMBER AMERICAN ASSOCIATION FOR THE
ADVANCEMENT OF SCIENCE; CHARTER MEMBER
AMERICAN INSTITUTE OF CRIMINAL LAW AND
CRIMINOLOGY; MEMBER OF THE AUTHOR'S
CLUB, LONDON.
AUTHOR OF THE FOLLOWING BOOKS: "THE IRREGULARITIES OF THE TEETH AND THEIR TREATMENT;"
"CHART OF TYPICAL FORMS OF CONSTITUTIONAL IRREGULARITIES OF THE TEETH;" THE
ETIOLOGY OF OSSEOUS DEFORMITIES OF THE HEAD, FACE, JAWS AND TEETH;"
"DEGENERACY: ITS SIGNS, CAUSES AND RESULTS;" "DEVELOPMENTAL
PATHOLOGY: A STUDY IN DEGENERATIVE EVOLUTION," ETC., ETC.

WITH 102 ILLUSTRATIONS

TOLEDO, O.
THE RANSOM & RANDOLPH CO.
1913

C. Franklin MacDonald. D.D.

THE PUBLISHERS' PRESS
CHICAGO

To

MISS ELLA CLAY

My Late Faithful Assistant

Whose scientific ability was recognized and appreciated by scientists

This work is dedicated

PREFACE.

In this book the etiology, pathology and treatment of peridontal disease have been worked out by actual research which covered a period of thirty-five years. Up to the present time teachers and practitioners have taught that disease of the gums and alveolar process was of a pyorrhœic or infectious nature. All etiologic experiments and treatment are made from this basis. The tendency at the present time is to study diseases of the body from a bacteriological view point. It is now known that many different tissues and organs of the body undergo physiologic and chemic changes which are often due to irritations setting up inflammation and may later under certain conditions become infected by bacteria. Inflammations of the mucous membranes due to acid states and burns are of this nature. It has been the aim of the author to classify the inflammations due to infections and those due to chemic changes and local and constitutional irritations. The diseases of the alveolar process due to infections are usually of systemic origin and develop symptoms in other parts of the body such as tuberculosis, typhoid fever, anthrax, actinomycosis, etc., and are rarely referred to the specialist for treatment. The patients referred to the specialists are those with inflammations of the gums, mucous membrane and alveolar process due to irritations and which may or may not later become infected by bacteria.

The author has observed that every vertebrate having two sets of teeth during life may possess an inflammatory condition of the alveolar process to a greater or less extent after the first set of teeth has developed depending upon environment and the condition of the system.

The pyorrhœic stage which may develop later is observed in only a very small per cent of patients. Happily, to a certain

extent, the profession has come to realize the importance of the two periods and the teaching of the pathology and bacteriology of the disease is much simplified and better understood.

The great law of medical science, that to know the cause is half the treatment, is as applicable to diseases of the mouth as to any other specialty of medicine, and is as applicable to dental problems as to those of biology generally. Treatment of any disease without knowledge of its pathology is practically a failure.

While much has been written upon the pyorrhœic stage of interstitial gingivitis and its treatment, during the past two decades, no new principle has been advanced whereby the parts can be restored to a healthy condition, or whereby the disease can be prevented. The disease is admittedly on the increase. This seems at first sight to indicate that dental prophylaxis and treatment, so far as this disease is concerned, are failures.

Nearly three decades ago I felt and expressed the necessity for more extended study (Dental Cosmos, 1886, page 689) of the clinical aspects and etiology of this disease. Even during apparently diverse and separated studies, such as those related to dental and maxillary irregularities and degeneracy, the necessity for this has forced itself still further upon me. In the present study, the disease has, of necessity, been considered from the broad standpoint of general pathology. In all instances where possible personal elements of error are present, these have been eliminated by having researches made by more than one observer.

The attempt has been made to summarize all researches on the subject. So much have opinions been intermingled that it is possible that proper credit for priority has unintentionally not been given.

The author is under obligation to the following scientists for their kind assistance: Dr. Ludwig Hektoen, Pathologist, Rush Medical College; Dr. Jerome H. Salisbury, Chemist, Rush Medical College; Dr. W. A. Evans, Pathologist, Columbus Medical Laboratory, Professor of Pathology, Chicago College of Physicians and Surgeons and Milwaukee Medical College; Dr. J. A. Wesener, Chemist, Columbus Medical Laboratory, Profes-

sor of Chemistry, Chicago College of Physicians and Surgeons; Dr. Vida A. Latham, Pathologist, Northwestern Uinversity, Woman's Medical School; Dr. Maximilian Herzog, Pathologist, Chicago Polyclinic Hospital; Professor Theo. A. Edwin Klebs and Dr. Robert F. Zeit, Pathologists, and Dr. W. L. Baum, Professor of Diseases of the Skin, Post-Graduate Medical School, Chicago; Dr. G. V. I. Brown, Professor of Oral Surgery, Dental Department, Milwaukee Medical College; Dr. Frederick Noyes, Histologist, Dental Department, Northwestern University; Dr. J. G. Kiernan, and to Blomgren Bros. & Co. for electrotypes, etc.

THE AUTHOR.

31 North State Street, Chicago.

CONTENTS

INTERSTITIAL GINGIVITIS

CHAPTER I.

HISTORY.

Inflammation of the peridental membrane and alveolar process is probably coeval with man. Some of the skulls found earliest in the cave-dwelling period exhibit evidences of its presence. In some of these, careful observation has shown deposits encroaching upon the roots of the teeth and resultant absorption of the alveolar process.

In the Swiss lake-dwellings and in the earlier Irish crannoges of like construction and situation, skulls are found, which exhibit deposits of tartar, inflammation of the peridental membrane and absorption of the alveolar process. These skulls were those of primitive races in whom disease of the jaws and teeth is supposed to be absent or infrequent. In the skulls of the peoples exhibiting the highest civilization at the earliest period—those of the Accadians and Egyptians—similar inflammatory conditions are to be found. This, however, was to have been expected, to judge from the dental directions left among the medical records of these peoples. The Greeks, Syrians, Arabians, Dravidians and Aryans of India and the early Burmese all suffered from this disorder. In the museum at Constantinople are the skulls of soldiers who fought at a battle 328 B. C. One of these skulls has the anterior alveolar process entirely absorbed away. The roots of the right central, the right lateral and the left central incisors are exposed.

Inflammation of the peridental membranes and alveolar process, it will be evident, is, therefore, not a modern disease; not a disease confined either to civilized or primitive races, but one which attacked man early in his evolution. Like most diseases it has been chiefly discussed and analyzed during the past two centuries.

In 1740 H. A. Fauchard [1] (while recognizing the disease in all its essential features and describing its principal symptoms) advanced no theory as to its origin.

In 1778 M. Jourdain [2] advanced the opinion that the disease was of scorbutic origin.

In 1821 L. Kaecker [3] discussed the disorder in an essay on the devastations of the gums and alveolar processes.

In 1822 M. Joirac [4] (in a discussion of the disease), while advancing no theory as to its origin, called it "pyorrhœa inter-alveolo-dentaire."

In 1860 Marshall de Calve [5] advanced the opinion that the disorder was of hereditary origin.

In 1867 Magitot, discussing the disorder, advanced the opinion that the gum, being in all cases only attacked subsequently, is not the original seat of the lesion. In his opinion systemic disorders like gout, rheumatism, albuminuria, diabetes and anæmia had an influence.

Bonwill [6] during the same year expressed the opinion that the disorder was due to thinness of the alveolar process between the teeth, thus depriving the peridental membrane and gum tissue of proper support. The want of proper articulation of the teeth also exerted an influence.

In 1870 Brown ascribed the disorder to serumal calculus.

In 1875 John T. Riggs, after whom the disorder is frequently called, entitled it (in a paper read before the American Academy of Dental Surgery) suppurative inflammation of the gums and absorption of the gums and alveolar process.

During the same year Scheff [7] of Vienna entitled the disorder periostitis dentalis. He was of the opinion that it originated from external irritation through mechanical, thermic and chemic changes. The real origin of the disorder was, in his opinion, very often obscure. He doubted, however, the influence of rheumatism.

[1] Independent Dental Journal, 1875.
[2] Philadelphia Journal of Medical and Physical Science, 1821.
[3] International Dental Journal, Vol. XIII.
[4] Journal of the American Medical Association.
[5] Journal of the American Medical Association.
[6] Dental Cosmos, Vol. XXIV.
[7] Wiener Med. Presse, Vol. XVI.

In 1876 Sirletti[8] (in a discussion of the pathology of the disorder, which he called alveolo-dental periostitis) regarded it as due to constitutional conditions, like rheumatism, scrofula, syphilis, etc., with local causes as exciting factors.

In 1877 Rehwinkle,[9] in a paper on pyorrhœa alveolaris, after citing from Albright (of Berlin) the claim that the disorder was due to uncleanliness, mercury and the suppression of habitual secretions, expressed the opinion that acquired and inherited constitutional defect often played an important part as etiologic factors. He was also of opinion that mercury exerted an influence in its causation. Salivary deposits were, in his opinion, without influence.

Clowes[10] was of opinion in 1879 that the general cause was lack of nutrition in the parts. The use of wedges often excited the disorder.

C. J. Essig,[11] in 1880, expressed the opinion that its predisposing causes were unknown, that it occurred as a rule in healthy persons, and that irregular and crowded teeth acted as an exciting cause.

In another paper during the same year G. A. Mills[12] expressed the opinion that the disorder was of systemic origin. Various mental and physical influences aided its progress, such as nervous exhaustion and bodily and mental overwork. In his opinion it frequently occurred in children and adolescents from eruptive fevers. The deposit was only a local manifestation of the disorder.

In 1881 N. S. Niles[13] expressed the opinion that constitutional conditions were, as a rule, without influence, and that local irritating deposits were the cause in twenty-five per cent of the cases coming under his observation. He was of opinion also that the amount of lime salts taken into the system in drinking water exerted an influence. A calcic and phosphatic diathesis had an influence in the production of the disorder.

---

[8] Gazzetta Medica di Roma, 1876.
[9] Dental Cosmos, Vol. XIX.
[10] Ibid., Vol. XXI.
[11] Dental Cosmos, Vol. XXI.
[12] Ibid., Vol. XXIII.
[13] Ibid., Vol. XXIV.

In 1881 (when there seemed to have been many contributions to the literature of the subject) Atkinson[14] expressed the opinion that nervous debility or original defect in innervation exerted an influence in the production of the disorder. The deposits of tartar were a secondary consequence. In the course of his paper he cited the opinion of Hamilton Cartwright that Riggs's disease commenced in an unhealthy condition of the gums with a secondary deposit of tartar.

In a paper read before the Dental Section of the International Medical Congress, Walker[15] claimed that the starting point of the disease was subacute inflammation passing into the depths of the alveolar process adjacent to the inflamed gum. In the discussion of this paper, Archovy and Joseph Izklai, of Buda Pesth, ascribed the disorder to minute organisms. Oakley Coles thought that systemic states were the predisposing factors, while minute organisms exerted an exciting influence.

In 1882, L. C. Ingersoll[16] regarded sanguinary calculus as a manifestation of the disorder, and distinguished it from salivary deposits.

Malasses and Gallippe,[17] in 1884, expressed the opinion that the disorder was of microbic origin.

In 1885, A. O. Rawls[18] expressed the opinion that the causes were environment with morbid factors, such as malaria, excessive sodium, etc., chloride and mercury.

In 1886, Reese[19] expressed the opinion that the disorder had its source in the uric acid diathesis resultant on abuse of alcohol.

During the same year, J. D. Patterson[20] expressed the opinion that the disorder was of catarrhal origin. Later, in 1886, J. N. Farrar[21] regarded the disorder as a combined result of systemic tendencies and local irritants. There was a peculiar condition of the system associated with hypersecretion laden with increased earthly deposits.

---

[14] Dental Cosmos, Vol. XXIV.
[15] Transactions of the International Medical Congress, 1881.
[16] Dental Cosmos, Vol. XXV.
[17] Ibid., Vol. XXVI.
[18] Ibid., Vol. XXVII.
[19] Independent Practitioner, Vol. VI.
[20] Dental Cosmos, Vol. XXI.
[21] Independent Practitioner, Vol. VII.

A. R. Starr later also expressed the same opinion. He, however, was unable to determine the local irritation factor, but regarded it as the same as that which causes exostosis of the cementum. He had found most cases in the upper jaws.

Black [22] designated the disorder phagadenic pericementitis. It was a specific infection of an inflammatory character, having its origin in the gingiva, and was accompanied with destruction of the peridental membrane and alveolar walls.

E. S. Talbot [23] during the same year regarded the disorder as a local one, due to both local and constitutional causes. The disorder began with simple inflammation of the gums, which afterward became chronic.

He laid particular stress upon the anatomy, physiology and pathology of the parts involved, there being no other structures in the human body like them, hence their easy susceptibility to disease.

We may consider, then, as predisposing and exciting causes a perverted condition of the secretions, low vitality of patient or tissues or both, calcic deposits and all diseases which affect the circulation such as drugs and auto intoxication, and as among the local causes catarrh, fistulae, salivary calculus, irritation from foreign substances, which are included as modern dentistry, such as detached bristles from the tooth brush, too great friction in brushing, injudicious use of the toothpick, the use of ligatures and regulating apparatus, application of the rubber dam and clamps, artificial dentures and regulating plates, accumulation and decomposition of food under artificial dentures, and at the necks of the teeth, drugs which over-stimulate the parts, the use of tobacco, fillings extending beyond the cervical margins, digestive derangements, contagion from unclean instruments, and improper mouth washes and tooth powders, especially charcoal. In a word, whatever irritates the gums, alveolar process and peridental membrane is likely to produce the lesion under consideration. The devitalization of pulps and the filling of roots, which throw increased work upon the mem-

---

[22] American System of Dentistry.
[23] Dental Cosmos, Vol. XXVIII.

brane, are also to be accounted as among the factors responsible for this pathological condition.

Talbot pointed out that the disease is contagious so far as one tooth becomes infected from another in the same mouth.

Of the status of this disease at the close of the year 1887, the following analytic picture was drawn by W. X. Sudduth[24]: "Pyorrhœa alveolaris is a term applied to the secondary stages of a disease that has its inception in a catarrhal stomatitis. Being confined, as a rule, to the margin of the gums surrounding the teeth, it might be called a 'gingivitis,' were it not for the general catarrhal tendency shown by the entire mucous membrane of the mouth and nasal passages. The intimate relation between a general catarrhal idiosyncrasy and pyorrhœa alveolaris is more than mere coincidence." Its common occurrence in persons who have irregular teeth has also been often noted by Dr. Sudduth, who considers that this fact has, besides the matter of uncleanliness, a direct bearing upon its pathogeny. It is well known that the irregularities of the teeth present an indication of a degenerative taint, and that persons in whom irregularities occur are very prone to catarrhal affections of the respiratory organs, including the nasal passage. Their skin is usually very susceptible to inflammatory affections. Another feature is the offensive ordor of the saliva of individuals who show this particular tendency to catarrhal affections even in persons who take most scrupulous care of the teeth. In the majority of cases, pyorrhœa is a stomatitis in which the local and constitutional factors in the production of the disease are largely dependent upon hereditary catarrhal dyscrasia for their ability to engraft themselves upon the tissues. This position is borne out by the clinical experience of Patterson, of Kansas City, Missouri, who reports thirty-eight cases of well marked pyorrhœa observed by him, thirty-three of which presented undoubted evidence of nasal catarrhal conditions; two were the result of direct irritation of misfitting partial plates, and the remaining three were apparently caused by calcific deposits. Patterson remarks that a close examination of the history of the above quoted cases

---

[24] Sajous' Annual, 1888, Vol. III, page 365.

confirms the opinion that the disease is, as a rule, an "oral catarrh."

From the foregoing W. X. Sudduth feels justified in classing the disease as a localized catarrhal stomatitis which may be either acute or chronic. Acute catarrhal inflammation of the gums begins in circumscribed points which present a bright or rose-red color, and which are generally located on the margin or the rugæ of the palate. There is but little swelling because of the dense nature of the sub-epithelial connective tissues. The gums present the same stages as are found in inflammation of other mucous surfaces—first dryness, followed by an increased secretion of mucus. The parts are very sensitive to pressure; the patient complains of an annoying, burning sensation. The appearance of the gums is noticeably smooth and glistening. They bleed easily when the brush is used or even during a meal. This stage does not last very long, but soon heals by resolution or passes into a chronic catarrhal stomatitis in which condition the gums become markedly swollen and turgid. They present a condition of tumefaction that sometimes rapidly passes into hypertrophy; at other times there is an indurated appearance that may last for some time. Granulation tissue may be produced as the result of overstimulation. The gums become detached from the necks of the teeth; and pockets are formed from which a fetid discharge may be pressed, giving a peculiarly disagreeable odor. Bullæ are apt to form, which, by rupturing in the process of mastication, give rise to intense smarting. The tongue constantly seeks the surface if the bullæ are on the inner side.

The pathologic changes which take place are, according to Newland Pedley, of England, "hypertrophy of the muco-periosteal fold around the teeth, accompanied by dilatation of capillary loops, enlargement of the papillæ and rapid proliferation of epithelial cells. Later the gums become firm and contracted and display increase of fibrous tissue. The changes which go on in the socket have not been as yet satisfactorily worked out. The examination of the jaws of carnivora, apparently affected with pyorrhœa alveolaris, leads to the supposition that osteitis of the alveolar process spreading toward the apex of the socket is pres-

ent. Later the alveolar walls become absorbed and are at times more or less denuded, while the fangs of the teeth become coated with a layer of thin, hard, green-brown tartar. Ultimately the disease progressing, the teeth, one after another, drop out."

From what has been said it will be seen that the pathology of pyorrhœa alveolaris may be explained in several ways. The general causes are local or symptomatic, or both combined. The most common cause of catarrhal gingivitis is found in local irritation, combined with some hereditary disposition to catarrhal affections. The next greatest etiologic factor is symptomatic—the local manifestation of a constitutional vice. The most common manifestation is that of syphilis and of its antidotes, mercury and potassium iodide, both of which sometimes find expression in a localized inflammation which may be the starting point for pyorrhœa alveolaris. As a complication of the disease in its secondary stages there can be no doubt of the action of micro-organisms, but Sudduth does not feel justified in conceding to them a position of specificity.

His position above quoted is sustained by Pedley, who finds that in most instances it is due to some constitutional condition. The fact that it is often symmetrical and frequently hereditary gives support as to this view. It occurs in mouths of patients whose health has been undermined by debilitating influences and injudicious habits of living. It is a common sequel of malarial fever. Young persons recovering from eruptive fevers are sometimes subjects of pyorrhœa alveolaris. Frequent pregnancies are a fruitful source of the disorder. Attention has been lately drawn to the shedding of the teeth in tabes dorsalis, but it does not, however, seem to be a constant symptom. Pedley's view, although tending entirely toward the constitutional character of the disease, does not militate against its catarrhal nature.

Bland Sutton has found shedding of teeth frequent in rheumatoid arthritis in animals. He has also met with it in mollities ossium and other wasting diseases. Magitot (who views the alveolar dental periosteum as a ligament and not of the same nature as osseous periosteum) calls the disease symptomatic alveolar-arthritis, and mentions especially as causes, chronic

Bright's disease and glycosuria, in which latter condition the phenomenon is absolutely constant.

Patterson holds that "mouth-breathing has, in his experience, been a very common accompanying condition which he cannot help connecting with the production of the disease. By it the gums are kept dry, their functions destroyed and the way paved for catarrhal inflammation. The majority of the patients he has been called upon to treat have been otherwise healthy, robust persons. From this fact he does not favor the idea of the disease being dependent upon constitutional derangement. It is, however, a well-known fact that these are the very class of people who when irritation is once set up in their system, present the most aggravated cases, by reason of their superfluous vitality. He says he has occasionally met with cases where the local condition was evidently aggravated by constitutional derangement and cure was thereby retarded. The great majority of cases, however, have shown no indication of constitutional predisposition, but have pointed unerringly to local irritation by means of which the function of the mucous membrane had been destroyed."

Syphilis and other affections may engraft themselves upon the gums without a predisposition of the parts toward an inflammatory condition, and having disturbed the normal status of the gingival margins they pave the way for subsequent disease in the alveolus. Certain drugs, such as mercury, phosphorus, lead, etc., have a known deleterious action upon the ligamentous attachment of the teeth.

In 1890 Miller [25] expressed the opinion that the disorder was of a parasitic nature.

In 1892 C. N. Pierce [26] charged the disorder chiefly to systemic predisposition and enthusiastically advocated the theory of Reese as to the influence of the uric acid diathesis.

In 1894 W. X. Sudduth [27] strongly urged the influence of lactic acid as a local factor in the disorder.

M. L. Rhein [28] presented a method of classifying pyorrhœa

---

[25] Micro-Organisms of the Human Mouth.
[26] International Dental Association, August, 1894.
[27] Ibid., Vol. XIV.
[28] The American Dental Association, August, 1894.

alveolaris in the following manner: "This classification is made by prefixing to pyorrhœa an adjective stating the name of the disease which is causing the pathological symptoms in the oral cavity as 'gouty pyorrhœa,' 'diabetic pyorrhœa,' etc."

The author,[29] after further researches, makes the positive statement that the etiology of the disease is due to both constitutional and local causes. He again reiterates and makes the positive statement that modern dentistry is producing more "pyorrhœa" (interstitial gingivitis) than any other one cause.

He again lays stress on the anatomy and physiology of the parts involved and that the alveolar process is a transient bony structure, simply for the purpose of holding the teeth in place after they have erupted; that the gums are rarely found in a healthy condition; that the peridental membrane is never invaded by pus germs so long as it is in a perfectly normal state; that in phthisical patients and those with low vitality and patients who have been ill for any length of time, a low form of inflammation of the gums extending to the peridental membrane and alveolar process with pus infection takes place; that the granular debris or calcic deposits in all cases are secondary considerations in the breaking down of tissue.

Neurotics and degenerates, whether wealthy persons or those confined in institutions, are mostly afflicted. Children as well as grown people suffer with the disease.

In 1897 the author laid particular stress on the poisons circulating in the blood causing interstitial gingivitis from autointoxication.

In 1899 he [30] he spoke of the possibility of calcic deposits on the roots of the teeth being "calcareous matter absorbed from the alveolar process in the immediate vicinity of the root," etc. This theory was later confirmed by researches on the alveolar process and mentioned in a paper on "Interstitial Gingivitis," published in The Dental Summary, 1903.

John Fitzgerald,[31] in 1899, claimed that "The production of pyorrhœa depends upon two factors, a predisposing cause and

[29] The International Dental Journal, April, 1896.
[30] Interstitial Gingivitis or So-called Pyorrhœa Alveolaris, page 169.
[31] Clinical Journal, March 1, 1899.

a local irritation. The predisposing cause may be tuberculosis, syphilis, scurvy, the exhaustion of acute infectious diseases or any other source of malnutrition. The exciting cause may be, and most usually is, a gingivitis produced in one of the ways to be presently described. There is also a pyorrhœa of gouty origin, in which the local necrosis of the peridental membrane is caused by gouty disease of one of the blood vessels in its substance.''

The views on etiology of this condition have varied, it will be observed, from purely constitutional causes to purely local causes, inclusive of microbic affections. In the main it will be obvious, however, that both constitutional causes, whether inherited or acquired, have been regarded as of influence by the majority of those who have written on the subject. There has, however, been very little exact study of either the predisposing or the exciting causes of the condition. Even the impetus given the study of etiology by bacteriology and embryology has as yet failed to make itself felt to any considerable degree in this department of dental pathology.

In 1903 the author spoke of the alveolar process as being an end organ; he also laid stress upon inflammation of the alveolar process as one of the first diagnostic symptoms in constitutional diseases in the same year.

In 1904 he spoke of the pulp as being one of the most perfect end organs in the body.

In an article, ''Endarteritis Obliterans and Arterial Hypertrophy in the Alveolar Process,''[32] the author first called attention to the calcic deposits (not tartar) on the roots of the teeth and stated that it was ''the detritus from the alveolar process.'' In this article, he lays emphasis on the fact that the alveolar process is not only a transitory structure but is also an end organ which makes it very susceptible to disease. The nerves and blood vessels approach a blank wall. The roots of the teeth, so far as disease is concerned, are foreign bodies.

Examination of the alveolar process of animals or human suffering from disease, in which the eliminating organs do not

---

[32] The Dental Digest, October, 1903.

throw off the effete matter (autointoxication), especially in syphilitic, tuberculous and scorbutic persons, easily reveals this morbid state.

In "Pathology of Root Absorption and Alveolar Abscess" [33] absorption of the alveolar process is always resultant upon irritation and inflammation. The strong point is emphasized that the alveolar process is doubly transitory and also an end organ. He also lays stress on the fact that in the constructive stage of the alveolar process, at the third and fourth periods of stress, the bone will reproduce itself but after the person has obtained his growth, very little or no restoration can take place. He also states that neuroses or degeneracy in the child has much to do with the success of treatment and that autointoxication also plays a great part in final results.

In a paper on "Peridental Abscess," [34] in some of the lower vertebrates, there is a continuous succession of teeth called polyphyodontia. When one tooth has performed its function it disappears to give way to another. This continues throughout life. In all vertebrates, including man, where only two sets of teeth are developed, it is called dyphodontia. The alveolar process and teeth of these vertebrates, including man, have retained phylogenetic remnants of the physiologic processes of removing transitory structures. Should man live long enough, he would normally lose his second set of teeth by osteomalacia or juvenile or senile absorption depending on the age of the patient. This is a great factor in the transitory nature of the alveolar process.

The author first mentioned autointoxication in an article, "Autointoxication in Its Medical and Surgical Relations to the Jaws and Teeth." [35]

---

[33] The Dental Digest, 1904.
[34] The Dental Digest, June, 1903.
[35] Journal of the American Medical Association, 1897.

CHAPTER II.

INTRODUCTION.

The attempt has been made in the present work to reduce to order the chaotic notions as to etiology, pathology and treatment which, during the present century, have gathered around the morbid condition erroneously entitled Pyorrhœa Alveolaris. While even erroneous titles may have their meaning so fixed by usage that any danger from the error involved in the title may be practically nil, still this is not the case with the title just cited. It suggests erroneous etiology, since pyorrhœa implies that there must always be a flow of pus, and hence that the disease must always result from infection with pus microbes. It implies erroneous pathology and erroneous treatment for the same reason. This being the case, such a title is so dangerously misleading as to compel in the present stage of dental science its modified use as a term for a disease. With a view of clearing up this question at the outset by the use of a proper title, I have adopted as a designation for the condition hitherto known as pyorrhœa alveolaris, the term "Interstitial Gingivitis." To this designation (as to all other attempts to express within a small space an extended pathology, etiology, prognosis, and clinical aspects) there are some objections. The term interstitial is used by some pathologists in a limited obscure sense. By the mass of dental pathologists, surgeons, physicians, and by medical lexicographers, the term is employed in precisely the sense in which it is used in the present work. The English surgeon and lexicographer Quain defines interstitial as follows: "Interstitial (inter, between; and sto, I stand); relating to the interstices of an organ. The term is applied in physiology to the tissue which exists between the proper elements of any structure, namely, some form of connective tissue. In pathology the word is used in connection with absorption when a part is gradually removed without any obvious breaking off, and also to indicate the implication of the interstitial tissues in morbid processes or their infiltration with morbid products, as interstitial pneumonia, interstitial hepatitis."

The Encyclopædic Medical Dictionary of the American Foster, states that interstitial has three significations: First, it is applied to a condition disseminated through the substance of an organ or part, and to an inflammation affecting the connective tissue of an organ; second, it is also applied to that form of growth which consists in the interposition of new elements between old ones, instead of in addition to the surfaces; third, it is applied to pathologic processes occupying the space between the essential parts of an organ which constitute its proper tissue, and is then employed in a sense opposed to that of parenchymatous.

A glance at the illustrations demonstrates the validity of the application of the term interstitial (in the sense of Quain, Foster, and the other lexicographers) to the condition erroneously called pyorrhœa alveolaris.

I have adopted the term gingivitis for reasons which will be obvious at the first glance. The philologic objection may be made that in it Greek and Latin are yoked together. Practically this is no objection, since German, French, as well as English, medical authorities employ such terms of mixed origin. Indeed the French (Mailhol[1] for example) apply the term gingivitis to the very condition to which I have applied it. In addition, they add to it the specific term "expulsive," to designate "a form of recession of the gums, accompanied by alveolar osteoperiostitis, and the gradual expulsion of the tooth from its socket." Foster[2] suggests the substitution of the term ulitis as more philologically correct. The term gingivitis, however, has crept into such wide use, that it would be futile to attempt to displace it for merely philologic reasons.

The term "gingivitis," however, conveys the idea that the disease always begins at the margin and is confined to the gums themselves. Here again we have an erroneous conception of the pathology of the disease. Thus in the formation of an alveolar or peridental abscess, there is an inflammation preceding the pyorrhœic stage. This is an interstitial inflammation and the gum tissue is not involved. Again, in inflamma-

---

[1] Odontalgie.
[2] Foster, op. cit.

tion due to mercury, lead, copper, brass, drug, autointoxications and other poisonings and irritations, there is an interstitial inflammation of the alveolar process and again the gums are not involved until the disease proceeds to a later stage.

My researches have shown that inflammation may take place from irritants or poisons circulating in the blood at any locality between the gum margin and the apical end of the root of the tooth. This inflammation may terminate in healthy resolution or it may go on to abscess with a discharge through a small fistula upon the gum which afterwards may heal without pain to the patient and the gingival margin may not be involved.

I have shown microscopic slides with these pathologic conditions in almost every constitutional disease. An inflammation may take place at the end of the root due to irritation or death of the pulp. The gingival border is again not involved. Irritation with inflammation around the roots of teeth occur in gout, rheumatism, syphilis and many other constitutional diseases and the gum margin does not become diseased or the seat of the inflammation may be at any point on the root and extend to the gingival border.

In all these illustrations the parts are restored to health without pyorrhœa alveolaris or even in many cases gingivitis. It is in such pathologic conditions that the word "interstitial" is added to "gingivitis." By this term, then, we know just what is meant. In no other term now in use can we locate the inflammatory area.

The two terms I have employed convey a fairly correct idea of the pathologic process involved in both deep-seated and superficial inflammation, and do not imply erroneous views as to etiology, pathology, prognosis and treatment.

The pathologic conception adopted in the present work anent interstitial gingivitis is that the disorder is a local inflammatory condition of the gums, or alveolar process, or both, tending to accelerate their normal tendency to disappearance at certain periods of stress, or involution, of which involution the changes produced by old age are a type. In this early senility of the gums and alveolar process, for such it may be termed, two great types of causes play a part; the exciting and the pre-

disposing causes. The exciting causes may be purely local, or may be local expressions of constitutional states. Thus it will be shown that the influence of uric acid when present is exerted as a local irritant, and not as a constitutional factor—the theory urged so strongly by Pierce. The uric acid hypothesis, once very dominant in medicine, is now losing its force. The trend of medical opinion is to consider it one of the danger signals of autointoxication which assumes prominence because of its tendency to excite local irritation. It is but one of a number of local expressions of constitutional defect. This view of the influence of uric acid in etiology the present work will try to demonstrate. Prominent among etiologic factors which have to be reckoned with, are pathogenic germs. In the present work it will be shown by all laws of bacteriology (under which investigations must be conducted) that there is no specific germ which is capable of producing the disease itself, and furthermore, that the pyorrhœa stage of the disease is merely a complication due to pyogenic germ infection of the already diseased gums. The views of Galippe as to a specific organism will be shown to have failed of support by numerous control experiments described in the present work. As these have been conducted by different experimenters they are free from the personal elements of error which vitiate the researches of Galippe, who violated that canon of the laws of Koch which compels production of the disease by the alleged specific germ. One predisposing factor will be shown in the present work to be the nature of the structures affected. This in pathology is called local predisposition. The gums, alveolar process, etc., will be shown to be transitory structures, as well as end organs, in themselves predestined (as already stated) to certain changes at certain ages. By the influence of the disease, about to be discussed, these changes occur prematurely. The influence of the toxic agents (mercury, potassium iodide, etc.) will be shown to have been exerted constitutionally through the central nervous system, their local effects being a secondary consequence of this. The same will be shown to be the case with conditions like scurvy and autointoxication (where the constitutional factor is most prominent), and with the great neuroses (paretic dementia, locomotor ataxia,

etc.). Here, as in the toxic conditions, one great element considered is the influence of the constitutional conditions upon the nerves governing local blood supply and tissue waste and repair. These influences are significantly illustrated in the various processes described later which tear down and build up.

The influence of morbid heredity as a direct factor will be shown not to be great. The influence, however, of degeneracy expressing itself along the lines of least resistance will appear as an ominously important factor. Heredity here, as elsewhere, is a warning rather than a destiny.

The influence of the nervous system on the processes of growth and repair, which is called its trophic function, has been shown to play a part in both the etiology of the disease and in its progress. This function has received but little attention from dentists, albeit its influence has been recognized in dental pathology in connection with great neuroses like paretic dementia and locomotor ataxia, in which gum disorders occur, followed by loosening of the teeth. The pathology of the disease has been discussed in the light of established facts of general pathology which have been accepted by the leading dental investigators, and not merely from a hypothetic standpoint. The disease has been regarded as a local exaggeration of certain physiologic processes, accompanied by diminution of the intensity of others. In the study of this phase of the question, the latest researches of dental pathologists as well as original observation and experiment have been employed.

Among the many questions which the present treatise is believed to settle (so far as experimentation can) is the following: The question of the influence and nature of its etiology. It is shown that here, as elsewhere in biology, the etiology of morbid conditions has many phases; that in it exciting and predisposing causes have alike to be considered; that while causes may be constitutional in origin they very often exert their action locally; that the disease is not a product of civilization nor a product of any one etiologic factor; that there is no ground yet adduced for believing the disease to be specifically infectious and due to a germ of a specific nature; that in it the germ infection occurs as a consequence of existing disease, and is not the

cause of the morbid condition, but one of its stages: Pyorrhœa. The experiments made, as well as the pathologic and clinical data, have been obtained from many observers, so that as many control observations should be had as were necessary to eliminate personal elements of error inevitable upon original observation and research. In the pathology no statement is made which is not demonstrated by corroboratory data, including a photograph of the condition. The treatment has been based upon the pathology and etiology. Its central idea is that the human being must be regarded as something more than his mouth and teeth; hence the duty of the dental scientist is, like that of all medical scientists, best shown in a prophylactic direction.

CHAPTER III.

TRANSITORY STRUCTURES.

THE JAWS.

Because of man's advance in evolution and because of the local degeneracies thereon resultant, through the law of economy of growth whereby one structure is sacrificed for the benefit of the organism as a whole, the face, jaws, teeth, gums, alveolar process and peridental membrane, being variable structures, are predisposed to disease in their very order of evolution.

The jaws are growing smaller because large ones are not required. The structures are changing their shapes to adapt themselves to the new environment. Thus—instead of broad large jaws with low vaults; short, broad alveolar processes with plenty of blood supply and vitality to resist mastication; teeth short, with large bell crowns to give plenty of room between the roots for considerable thickness of the alveolar process for the nourishment of the peridental membrane and support and protection of the gum tissue—small narrow jaws occur with apparently high vaults; long, slender and thin alveolar processes, which are not used in mastication with sufficient force to carry the blood for the nourishment of the tissues. The teeth are changing their shape, causing the roots to come closer together, and thus lessening the area of the alveolar process.

That the jaws of man are growing smaller is easily demonstrated. Scientists claim two types of heads as a starting point in the study of head and face deviations, the brachycephalic (round) head of the Teutonic race and the dolichocephalic (long, narrow) head of the negro. These two primitive types of heads possess two distinct types of jaws. The brachycephalic head has a large round dental arch; the jaws and dental arches may or may not protrude, while the dolichocephalic head has large, long, protruding jaws and dental arches. The normal natural tendency in the evolution of man is to eventually (owing to

admixture of races and environment) harmonize these two types and produce a mesaticephalic (medium) head, face and jaws. The frontal development of the brain in phylogeny is gradually causing the skull bones to project forward. Owing to disuse in ontogeny the jaws are growing smaller and receding.

While these changes are gradually going on normally, certain factors are brought to bear upon the mother and child which increase or diminish the nutrition of the child and bring about arrest or excessive development of the face, jaws and teeth. These factors are an unstable nervous system [1] either in parent or child or both.

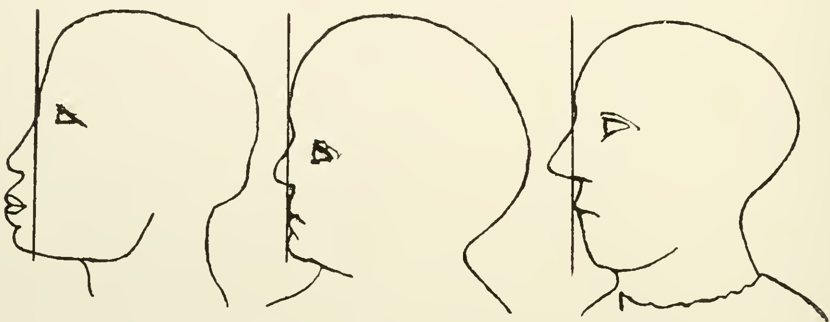


FIG. 1.—ILLUSTRATES THE PRIMITIVE HEAD WITH A RECEDING FOREHEAD AND PROTRUDING FACE AND JAW. THE SECOND ILLUSTRATION SHOWS AN ADVANCE IN EVOLUTION WITH THE HEAD, FACE AND JAWS ON A PERPENDICULAR LINE, WHILE THE THIRD SHOWS A RECESSION OF THE FACE AND JAWS.

That these views can be verified are easy of demonstration. Drop a perpendicular line (Fig. 1) from the supraorbital ridge below the lower jaw. It will be found that in most of the primitive races the jaws will protrude outside the line and the forehead will remain inside the line. As a human face has advanced in its phylogeny, the reverse has taken place.

Thus an examination of ten thousand people in the streets of London revealed the fact that in only four and thirteen one-hundredths per cent of people examined did the jaws extend outside the perpendicular line; twelve and eighty-seven one-hundredths per cent on the line, and eighty-three per cent inside the line. In an examination of three thousand English school children (about ten years of age) ninety-three per cent possessed jaws

[1] Talbot: Developmental Pathology: A Study in Degenerative Evolution.

inside the perpendicular line; six per cent on the line and one per cent outside the line. An examination of eight thousand people of Boston showed six per cent of jaws extending beyond the perpendicular line; fourteen per cent on the line and eighty per cent inside the line. The examination of the people of Boston was made because they more nearly represent those of England in this country in nationality, environment and influence of marriage and disease. It has required more than one thousand years to bring about these results. A more vivid illustration of this change and one that can be easily understood by the reader is that which has taken place in the negro in the more settled parts of America in two hundred and fifty years.

An examination of the lowest negro type in Mississippi was made for me by Dr. William Ernest Walker of New Orleans. His examinations of three hundred and fifty-seven showed the facial angle protruded beyond the perpendicular line in ninety-seven and five-tenths per cent of jaws, while two and five-tenths per cent of jaws examined were on the line. An examination by Dr. Arthur R. Dray of six hundred and eighty-six negroes in Philadelphia, eighty-three and fifty-seven one-hundredths were found outside the perpendicular line, fifteen and ninety-five one-hundredths on the line and forty-two one-hundredths inside the line. An examination of one thousand and eighty-five in Chicago, fifty-one and six one-hundredths per cent protruded; thirty-one and eight-tenths were on the line and sixteen and six-tenths per cent were inside the line. An examination of one thousand negroes in Boston by Dr. Eugene F. O'Neill showed forty-five and four-tenths per cent outside the line; thirty-nine and five-tenths per cent on the line and fifteen and one-tenth per cent inside the line. It will be seen, therefore, that in Northern and in old negro families, from race admixture and environment, there is less protrusion and more recession than in the Southern pure negroes. Arrest of the bones of the face is as common in old negro families in the North as among the Caucasic races.

To further substantiate this claim a comparison of the measurement from the outside of the first molars of the upper jaws of modern races with ancient skulls and ancient races may be here given. Examinations made by the late Dr. Mummery, in

1860, of ancient British skulls measured 2.12 inches, maximum 2.62 inches, with an average of 2.37 inches. The modern English jaws measure minimum 1.88 inches, maximum 2.44 inches with an average of 2.19 inches. The jaws of people living in America measure minimum 1.75 inches, maximum 2.52 inches with an average of 2.14 inches. The difference between the ancient Roman soldiers and modern Romans is the same as that of the English.[2] The lateral measurements of the pure negro as found in Mississippi are minimum 2.25 inches, maximum 2.75 inches, with an average of 2.51 inches. The lateral diameter of modern negroes varies considerably owing to neurasthenia in the parents and disease in the child. Some jaws measure as low as 1.75 inches. The jaws of modern negroes residing in Boston for many generations are not unlike those of the native whites.

A further demonstration that the jaws are becoming smaller is shown by the disappearance of the third molar, or the irregularties resultant on its eruption because of want of room, or its eruption with pain for like reason. In the primitive races it is large and well developed.

Dr. Charles Ward says a "point in which the jaws of aboriginal tribes, are, as a rule, superior to those of civilized races is in the proportion of the horizontal ramus. As pointed out by Harrison Allen, the alveolar and inferior border of the jaw tend to parallelism in savages, while in civilized races the symphysial height is usually greater than the height in the vicinity of the molars. This may be due to gradual degeneration of the platysma myoides muscle. Of the significance of the 'antegonium' or 'pregonium' of the same author I am uncertain, but incline to the belief that it is a 'stigma of degeneration.' Finally, an as yet incompleted study of the relative proportion of jaw to skull has convinced me that the jaws of savages are not only proportionately but actually heavier than our own, and that the 'cranio-mandibular index,' as I term it, which is the ratio between the weight of jaw and weight of cranium, rises steadily as we descend from semi-civilized to barbarous and savage tribes."

"Thus, while the white males examined gave an index (pro-

[2] Talbot: Irregularities of the Teeth.

portion of jaw to skull) of 11.8, the male Australians presented an index of 15.4.

"Absolute size of the lower jaw is greater in savages: Of nine aborigines, including seven North American Indians, one African and one American negro, six Malays and five Australians, all with beautifully perfect teeth, the mean weight of the jaw was 102.4 grams. Of eighteen white males the mean weight of the jaw was only 83.4 grams. Yet the weight of the skull was nearly alike in both classes, being 690.9 grams for the aborigines as against 680.5 for the whites. The weight of the lower jaw compared with that of the cranium, or the cranio-mandibular Index is 15.6 for aboriginal men as against 12.16 for white men. It is 46.2 for the anthropoid apes, our nearest living relatives among mammals."

The change in the two extremes of heads, the brachycephalic and the dolichocephalic to the mesaticephalic also produces change in the shape of jaws in like manner. Instead of the large round jaw of the brachycephalic and the long narrow jaw of the dolichocephalic, a medium size jaw development also follows.

CHAPTER IV.

TRANSITORY STRUCTURES.[1]

THE ALVEOLAR PROCESS.

The alveolar processes are situated upon the superior border of the inferior maxilla and upon the inferior border of the superior maxilla. These bones, considered a part of the maxillary bones often so described by anatomists, should, however, be considered from a more careful study of their physiology and pathology as practically distinct bones—their structure, functions and embryology differ so completely from the structure and functions of the maxillary bones. The superior and inferior maxillæ are (unlike the alveolar processes) composed of hard, compact bone structure. The large, powerful muscles attached to them indicate that powerful work is to be accomplished. When fully developed they retain their full size through life. The alveolar processes are composed of soft, spongy bone of a cancelloid structure. As early as the eleventh week of intrauterine life, calcification of the deciduous teeth commences, and by the twentieth week calcific material is abundantly deposited. Ossification is also rapidly progressing about the dental follicles. At birth, the sacs are nearly or quite inclosed in their soft, bony crypts, and the crowns of the teeth upon their outer surface are composed of enamel, which is dense and hard. The embryologic phases of the dental shelf elsewhere cited[2] indicate this development.

The alveolar process, being soft and spongy, molds itself about the sacs containing the crowns of the teeth and about their roots after their eruption, regardless of their position in the jaw. While the alveolar processes have grown rapidly, they have up to this time developed only sufficiently to cover and

---

[1] Pyorrhœa Alveolaris. Paper No. 2. The International Dental Journal, April, 1896.

[2] Talbot: Irregularities of the Teeth, page 93.

protect the follicles while calcification proceeds. When the crowns have become calcified and the roots have begun to take in their calcific material, absorption of the borders of the processes takes place in the order of the eruption of the teeth. When the teeth have erupted, the alveolar processes develop downward and upward with the teeth until they attain the depth of the roots of the teeth, which extend in most instances into the maxillary bones in the anterior part of the mouth at least, and the upper and lower teeth rest at a point in harmony with the rami. The depth at which they penetrate the bone differs in different mouths. This depends upon the length of the roots and the alveolar process. This in turn depends upon the length of the rami. The incisive fossa, the canine eminence and the canine fossa give evidence of this externally. These sockets are lined with extensions of the process, thus making its upper border irregular. The crypts of the permanent teeth are located at the apices of the roots of the temporary teeth. The permanent teeth have large crowns which touch each other, forming a line to the posterior part of the jaw. These teeth, as they erupt, entirely absorb the alveolar process which surrounded the temporary teeth, and as the new set comes into place a new process is built up around them for their support.

The process of absorption of the alveolar process and the building up of new bone around the first and second set of teeth is inflammatory. This then is the beginning of interstitial gingivitis in the life of every individual. Whether this primitive inflammation continues through life or not, will depend upon the general health of the person and his ability to keep his gums and alveolar process in a normal, healthy condition after the temporary teeth have erupted.

The permanent teeth require a deeper alveolar process to support their roots, which are much longer than those of the temporary teeth. Hence the difference in the depth of the vault of the first and second sets of teeth.

The alveolar process of each superior maxilla includes the tuberosity, and extends as far forward as the median line of the bone, where it articulates with the process upon the

opposite side. It is narrow in front, and gradually enlarges until it reaches the tuberosity, where it becomes rounded.

The process is composed of two plates of bones (Fig. 2), an outer and an inner, which are united at intervals by septa of cancellous tissue. These form the alveoli for the reception of the roots of the teeth. In some cases the buccal and labial surfaces of the roots of healthy teeth extend nearly or quite through the outer bony plate and are covered by the peridental and mucous membranes only.

This plate is continuous with the facial and zygomatic surfaces of the maxillary bone. The inner plate is thicker and stronger than the outer, and is fortified by the palate bones. The external plate is irregular upon the outer surface, promi-

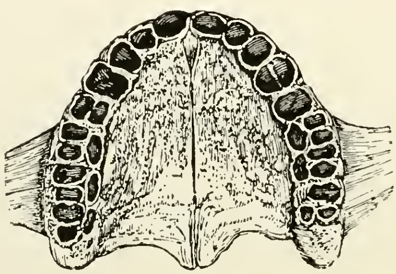


Fig. 2.—Diagram of the Superior Maxillary Bone with the Teeth Removed.

nent over the roots of the teeth, and depressed between the roots or interspaces.

With the change in the size of jaws there is also change in the shape of the vault and alveolar processes. When the dental arches are large, measuring from 2.25 to 2.50 inches, the vaults are low and the alveolar processes are short and thick, not only giving stability to the teeth, but also plenty of nourishment. Now that the dental arches are growing smaller, with an average of from 1.90 to 2.00 inches, the vaults are higher in proportion, the alveolar processes long and thin. This renders the teeth and jaws more susceptible to trophic changes and hence to disease. The alveolar process in the anterior part of the mouth, in which the incisors and cuspids are situated, is much thinner than in the posterior parts.

The sockets for the incisors and cuspids are conical and much larger than any of the other single sockets. The alveolar process is longer and thinner than at any of the other teeth. The sockets for the bicuspids are flattened upon their anterior and posterior surfaces, and near the apices they are frequently bifurcated. The sockets of the molars are large at the openings. About the middle of their length, however, they are divided into three smaller sockets for the reception of the roots. In the case of the third molar the number of sockets ranges from one large cavity to three or four of smaller size. When disease attacks the tissues, destruction is, therefore, more rapid in its progress in the anterior parts of the mouth than in the posterior, where the processes are thicker and more nourishment is required.

The septa are very thin at the margin and gradually increase in width to the middle of the jaw, where they become thicker, and are finally lost in the substance of the jaw. Some septa are thicker than others, and where two teeth are widely separated, the width of the septa naturally corresponds to the space between the teeth.

What is true in regard to a change in the size of the jaws is also true in respect to the shape of the crowns of the teeth. While they are not growing smaller in proportion to the size of the jaws, they are changing shapes. Once they were quite bell-shaped, giving considerable space between the roots for a thick alveolar process, thus rendering support to the peridental and mucous membranes, now the shape has changed. The proximal surfaces are almost straight, lessening the width and thus allowing only for a thin septum, with barely sufficient surface to support the tissues without material blood and nerve supply.

The sockets are lined with a thin plate of compact, bony substance, extending from the outer and inner plates of the alveolar process to the apex, where there are small openings for the entrance of the nerve and blood vessels for the nourishment of the teeth.

The bony plate has upon its inner surface the elastic peridental membrane, which acts as a cushion for the teeth, while it is surrounded by a spongy bone.

The teeth are held firmly in their alveolar sockets by the peridental membrane. Teeth with one conical root, and those with two or more perpendicular roots, are retained in position by an exact adaptation of the tissues. Teeth having more than one root and those bent or irregular, receive support from all sides by reason of their irregularity. Fig. 3 (a section of the jaw of a cat) illustrates the relative position of the teeth, peridental membrane and alveolar process to each other.

After the removal of the permanent teeth the alveolar process is entirely absorbed. Fig. 4 shows how the absorption takes place. The teeth have all been removed from the superior maxilla and the alveolar process has been entirely absorbed.

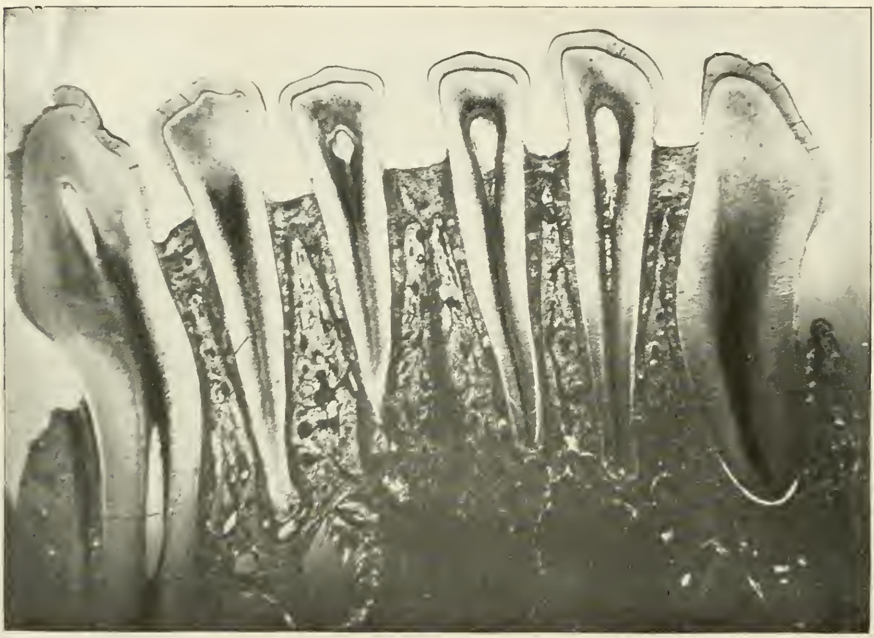


FIG. 3.—GROUND SECTION OF JAW AND TEETH OF CAT. (ANDREWS.)

The molars on the lower jaw having been extracted, absorption of the alveolar process has resulted in marked contrast with the anterior alveolar process, which remains intact and holds the teeth firmly in place. It is, hence, evident from the changes which occur, from the first development of the teeth to their final extraction, that the alveolar process exists solely to protect the teeth in their crypts during development and after

eruption. After the temporary teeth are in place the alveolar process remains unchanged (except by gradual enlargement in harmony with the growth of the maxillary bones) until about the sixth year, when the second set appears. The crowns of the permanent teeth require more space than those of the tem-

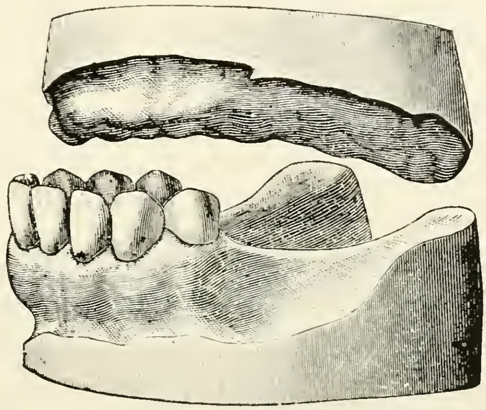


FIG. 4.—PLASTER CASTS OF THE SUPERIOR AND INFERIOR JAWS IN POSITION. ALL THE TEETH HAVE BEEN REMOVED ON THE UPPER JAW, AND THE MOLARS AND SECOND BICUSPIDS ON THE LOWER JAW. ABSORPTION OF THE JAWS WHERE TEETH HAVE BEEN REMOVED WELL ADVANCED.

porary set; and the alveolar process must necessarily enlarge to accommodate them. This enlargement of the alveolar process is caused chiefly by formation of the crowns of the permanent teeth before eruption, and to a limited extent only by

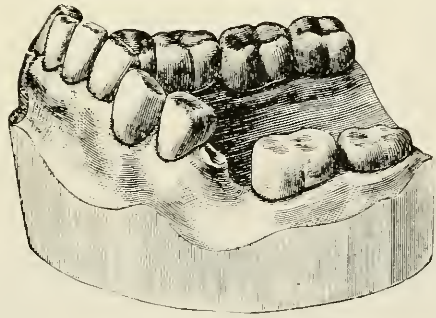


FIG. 5.—THE ANTERIOR ALVEOLAR PROCESS EXCESSIVELY DEVELOPED, CARRYING THE TEETH UPWARDS.

growth of the maxillary bones. These may cease development at any period of the life of the individual, or continue as late as the thirty-sixth year. As diameter of the crowns of the permanent teeth form a larger circle than that of the maxillary

bones, the alveolar process must necessarily increase its diameter and present large spaces between the roots of the teeth for the development of the alveolar process.

The process is solely for retaining the teeth, and if for any reason the dental follicles should not be present, and the tooth should not erupt, or if it should be extracted early, the process would not be developed at that point. In my collection of models are cases of arrested development of the alveolar process, caused by the lack of bicuspid and lateral incisor germs, and by extraction of deciduous and permanent teeth.

If one or more teeth were not to antagonize, the alveolar process would extend beyond the natural border, carrying the teeth with it. A marked illustration of this is seen where the molars are decayed to the gum and the roots remain. The vascularity of the process may be such that hypertrophy results. Excessive development of the alveolar process is frequently observed by every practitioner in connection with the anterior inferior teeth. When the articulation is normal, occlusion of these teeth never takes place. Frequently (especially in patients from six to twelve years of age) these teeth extend to and occlude with the mucous membrane of the hard palate. Such a case is illustrated in Fig. 5. This model is taken from the jaw of a person thirty-seven years of age, but this excessive development took place between the ages of six and twelve years, since at that period the vascularity of the tissues is more vigorous, and the development of the process more formative than at any period subsequent to the development of the first permanent teeth.

In one patient under observation the incisors and cuspids, together with their alveolar process, are situated upon the external surface, while the bicuspids, molars and their alveolar process are located upon the inner border of the jaw. In another patient, the alveolar process failed to cover the roots of the bicuspids and molars upon the outer surface, the teeth having forced themselves into a larger circle through the alveolar process by the contact of the crowns. The roots in this patient

---

[8] Dental Surgery, page 44.

can easily be outlined by the finger through the mucous membrane, the outer plate of the alveolar process barely, if at all, covering them. Tomes[3] illustrates a patient of faulty development of the outer plate of the alveolar process, exposing the crown of the temporary teeth. This occurred in a hydrocephalic. I have a number of models showing the anterior alveolar process projecting beyond the normal position through the forward movement of the molars. This may be due to a natural movement of the molars forward, or the process may be forced forward by the improper occlusion of the jaws. The teeth are moved from one position to another simply by the force consequent upon absorption and deposition of bone. This is noticeable in the spaces between the centrals, when the alveolar process develops to a larger circle than is necessary to accommodate the teeth. The alveolar processes are influenced in one direction or the other by the pressure of articulation. This results from inharmonious development of the jaws. The teeth may come together in such a manner as to throw the alveolar processes either to the right or the left, thus producing a full, round arch upon one side of the jaws, and a perfectly flat or straight arch upon the other. Occasionally both upper and lower alveolar processes are carried forward in the same manner. The alveolar process upon the lower jaw is more liable to be found upon the inner border of the jaw than is the upper alveolar process, as the inferior maxilla is larger and more dense than the superior, and when the teeth are once in position upon the lower jaw they are not so liable to subsequent change. Owing to this the teeth of the superior maxilla do not form so great a circle. This causes the teeth upon the sides of the jaw to conflict, and the lower teeth and alveolar processes to be carried in, while the anterior teeth of the lower jaw are held inside of the superior anterior teeth, thus carrying the alveolar processes inward.

The teeth are continually changing their positions in the mouth. This is as often beneficial as it is detrimental. That the teeth may perform their full function, they should not only remain firmly in the alveolar process, but they should also antagonize properly. The teeth may be compared to the bricks

in an arch. Remove a brick and the arch falls to pieces. It is frequently found that the teeth do not articulate properly; by a slight movement, or by cutting away the grinding surfaces, a better articulation may be secured. When this operation is performed, the teeth move in their sockets by absorption and deposition of bone, demonstrating the fact that the process changes in shape and substance. Ziegler[4] says absence of functional use is a frequent cause of premature lacunar absorption of the bone. This form of atrophy from disuse occurs not only when a limb or a part of a limb is deprived of its normal activity, but also when portions of a single bone cease to perform their function of support, and finally, like all the bones of the body, as age advances, normal or physiologic absorption takes place, while the teeth are still in the jaws. Unlike other bones of the

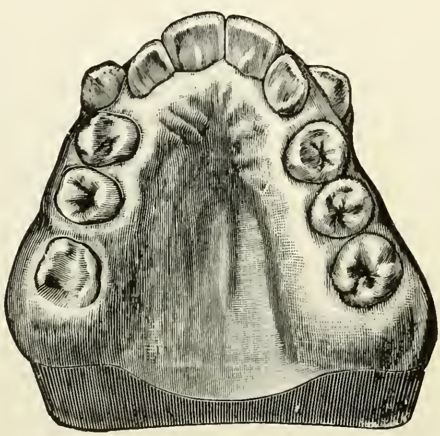


Fig. 6.—Hypertrophy of the Entire Alveolar Process.

body, however, the absorption of the alveolus progresses to a greater extent because of the unstable condition of the structures.

From what has already been said of the vascularity of the alveolar process, it is evident that hypertrophy of the tissue may ensue from an unbalanced nervous system and from simple irritation of varying degree. This unbalanced nervous system may act directly upon the pituitary body, producing hypophyscal disorders which in turn affect the growth of the jaws and alve-

[4] A Text-Book of Special Pathological Anatomy, page 145.

olar process. The irritation consequent upon the eruption of the teeth, together with the excessive blood supply, are both primal causes of overbuilding of tissue, i. e., hyperplasia.

The ragged roots of the temporary teeth, produced by absorption of the gases from the putrescent pulps, and the pressure of the permanent crowns against the tissues, produce sufficient stimulation to excite physiological action. Tissue-building generally is seen in connection with all the teeth, and the process becomes unnaturally thick, the teeth frequently are carried in one direction and another; cementosis of the roots of the teeth and hypertrophy of the process result.

In cases of hypertrophy of the alveolar process, enlargement is associated with the inner plate of the alveolar process. In patients coming under my observation the inner plate in

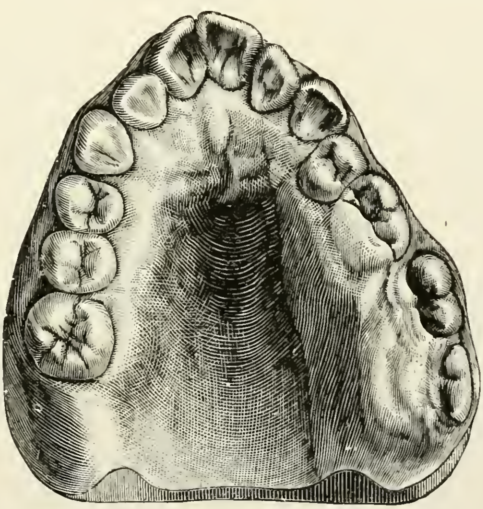


FIG. 7.—HYPERTROPHY OF THE ALVEOLAR PROCESS AROUND THE LEFT SUPERIOR MOLAR TEETH.

most is the part of the alveolar process affected (Fig. 6 case). The outer plate, although quite irregular from the arrangement of the teeth, is usually normal in thickness. This disparity in the two plates of the alveolar process is due to the fact that the inner plate of the alveolar process possesses a large blood supply, the posterior or descending palatine arteries furnishing the ossific material. I have observed but few

patients where hypertrophy has extended to and included the outer plate. When the outer plate becomes involved the alveolar process assumes a very thick condition. Occasionally hypertrophy will affect one side only or one distinct locality (Fig. 7). In this patient the enlargement is upon the left side and extends from the first bicuspid posterior to and including the maxillary tuberosity. Instead of the force being directed inward, as is generally the case, the process is forced outward and backward. This enlargement occurred previously to the development of the second and third molars. The alveolar process extends downward and occludes with the teeth upon the lower jaw, thus preventing the molars from erupting.

The causes which produce hypertrophy of the alveolar process are those due to an unstable nervous system. This subject is discussed in my work on "Developmental Pathology: A Study in Degenerative Evolution."

(Late researches have shown that the hypophysis and its disorders have much to do with arrest and excessive development of the body as a whole or of particular organs or structures of the body. The author many years ago called the attention of the profession to the fact that an unstable nervous system of the parent or disease in the child after birth produced excessive or arrest of development of the tissues. It is possible that the unstable nervous system of the parent or the diseases of the child act first upon the pituitary body, which in turn acts upon the tissues themselves or that they both act together.)

CHAPTER V.

THE ALVEOLAR PROCESS UNDER THE MICROSCOPE.

Under the microscope, two systems of Haversian canals are seen in the alveolar process. Kolliker[1] describes these as follows:

"The Haversian canals are of two kinds. One with the regular lamellæ system surrounding it, and the other, the so-called Volkmann's canals, containing the perforating vessels from Von Ebner, which have no surrounding lamellæ, but sim-

Fig. 8.—Section of Bone Showing Blood Vessels of Von Ebner (Kolliker).

ply penetrate through the layers of bone. Volkmann's canals are present in all tubular bones in old and young. While especially present in the outer basal lamellæ, they occur also in the interstitial leaflets and in the inner chief lamellæ as well as in the periosteal layers of the skull bone. Here their number is very variable (Fig. 8). They run partly transversely or obliquely, and also partly longitudinally, through the lamellæ.

[1] Handbuch der Gewebelehre, page 272.

Many of these canals open in the outer or inner surfaces of the substantia (compact substance), and also here and there in the Haversian canals, and form altogether usually a wide-meshed irregular network. In their structure they are sometimes smooth and sometimes furnished with dilatations and angles projecting in and out in profile. The widest has a diameter of 100 micrometers or more, and the narrowest not more than 10 or 20 micrometers, and there are still narrower ones which are altogether obliterated, appearing like rings or circular-formed

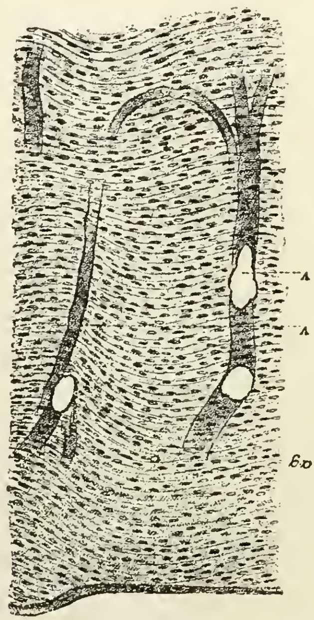
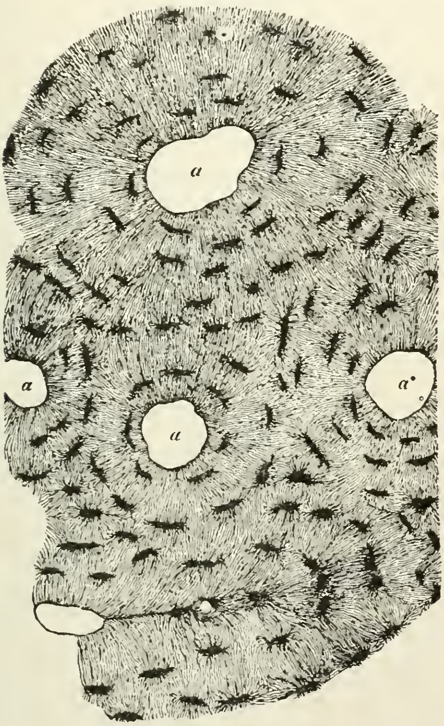


Fig. 9.—Section of Bone (Higher Magnification) Showing Blood Vessels of Von Ebner.

structures without any lumen, or like those far from rare obliterated true Haversian canals first described by Tomes and de Morgan. The contents of the Volkmann canals are the same as the Haversian canals."

Fig. 8 is a cross section of the medulla of a calcified human humerus slightly changed. The outer lamellæ contains a large number of Volkmann's canals running longitudinally and transversely and extending through the outer plate of bone into the periosteum. Fig. 9, the cross section of the section seen in

Fig. 8, shows these canals more highly magnified. The Haversian canals are large round spaces (Fig. 10), containing a single artery and vein. The fine hair-like spaces running from these large spaces are the canaliculi. The dark spots circulating each Haversian canal are the lacunæ. The canaliculi run from one lacunæ to another or into a Haversian canal or they anastomose with each other. The rings of bone about each Haversian canal are called lamellæ. The lacunæ seem to be about uniformly distributed throughout the bone. The spaces



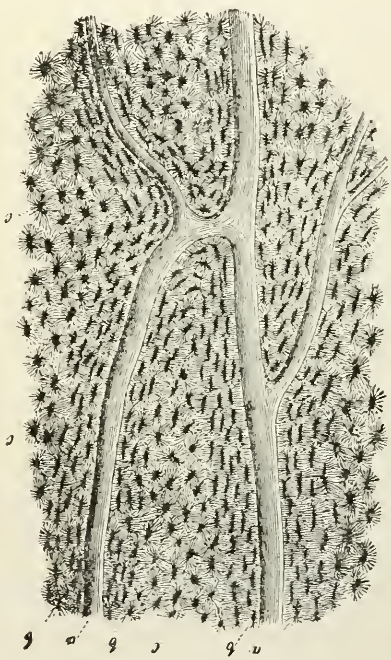


FIG. 10.—TRANSVERSE SECTION OF THE DIAPHYSIS OF THE HUMERUS MAGNIFIED 350 TIMES.
A, HAVERSIAN CANAL. DARK SPACES, LACUNAR.
HAIR-LIKE SPACES, CANALICULI.
FIG. 11.—LONGITUDINAL SECTION OF BONE MAGNIFIED 100 TIMES.

between the lacunæ and canaliculi are filled with lime salts.

A longitudinal section of bone (Fig. 11) is similar in appearance to the cross section. Instead of the lacunæ being arranged in rows around the Haversian canals they are parallel. It will be noticed that the Haversian canals run in different directions and communicate with each other at certain intervals. The foregoing description, with illustrations from Kolliker, is essentially that of the minute anatomy of the alveolar process.

CHAPTER VI.

THE GUMS, PERIOSTEUM, MUCOUS AND PERIDENTAL MEMBRANES UNDER THE MICROSCOPE.

GUMS AND MUCOUS MEMBRANE.

The tooth, according to Minot,[1] is a papilla which projects into the epidermis, and ossifying in a particular way, changes into ivory around the soft core or pulp. To the papilla the epidermis adds a layer of enamel. The tooth proper unites with a small plate of dermal bone at its base. By a modification on the jaw, the epidermis first grows into the dermis, and then the dermal tooth papilla is developed. The teeth were primitively organs of the skin and widely developed over the surfaces of the body. As the mucous membrane is practically a continuation of the skin, it, in accordance with the law of individuation, became specialized and lost some of the functions of the skin while developing the others to greater perfection.

The mucous membrane lines the cavity of the mouth, the nose, and extends through the larynx into the lungs and through the œsophagus into the stomach. It covers the tongue, jaws, alveolar process, dipping down between the necks of the teeth and the alveolar process as far as the peridental membrane, leaving a free space between the membrane and the teeth through its entire length.

It consists of two layers (Fig. 12), the epithelium (A) and corium (B), separated by the basement membrane (C). The epithelium is composed of the epithelial cells: First, one row of columnar cells (D) situated upon the basement membrane (C); second, two rows of six-sided prickle shells (E); third, two rows of six-sided cells (F); fourth, two or three rows of squamous cells (G); and fifth, four or five rows of flattened dead cells (H), which were originally the columnar cells upon the basement

[1] Embryology, page 481.

membrane. The young new cells are the columnar cells which pass from one stage to another, changing their shapes until they eventually become dead cells and are exfoliated from the surface of the tissue. The basement membrane (C) is made up of fibers running longitudinally, from papillæ, which allow the tunica propria containing blood vessels and nerves to pass up into the epithelium structure.

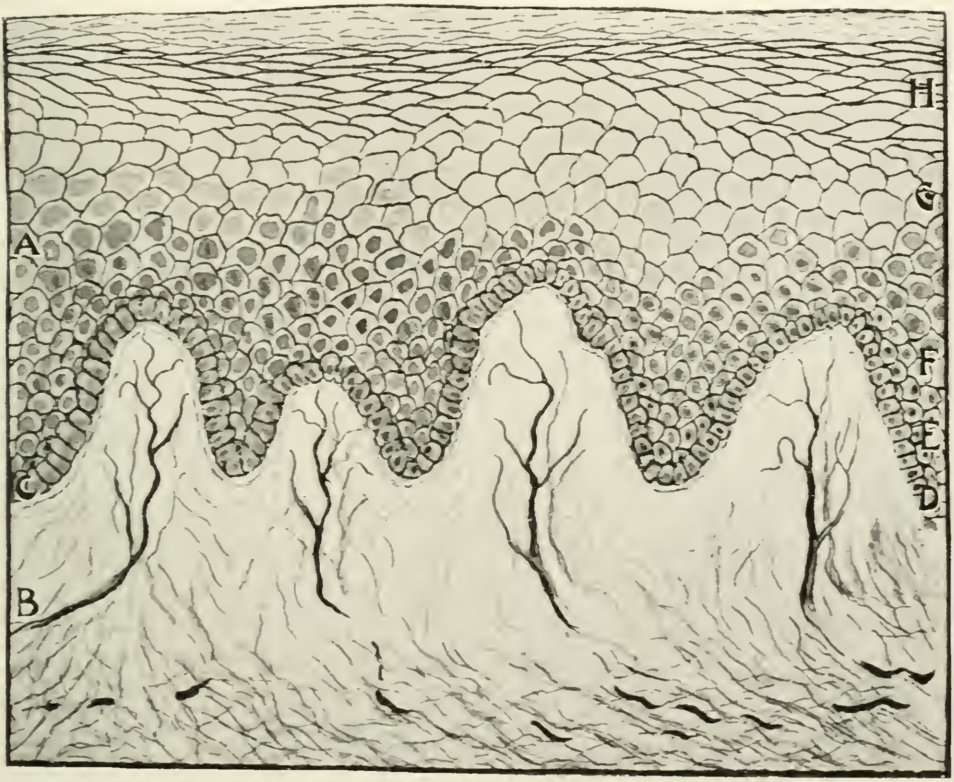


FIG. 12.—DIAGRAMMATIC ILLUSTRATION OF THE EPITHELIUM AND SUBMUCOUS LAYERS OF THE MUCOUS MEMBRANE.

A, Epithelium. B, Corium. C, Basement Membrane. D, Columna Cells. E, Prickle Cells. F, Six-sided Cells. G, Squamous Cells. H, Flattened Dead Cells.

The corium (B) (which lies below the basement membrane) is composed of alveolar connective tissue, white yellow fibrous connective tissue, muscular fibers, nerves, blood vessels and lymphatics. It is made up of the tunica propria and the submucosa.

The tunica propria (beautifully shown in Fig. 13) consists

of interlacing connecting fibers interspersed with much elastic fibrous tissue. This tissue penetrates the epithelial layer in the form of cone-shaped papillæ, varying in length with the thickness of the epithelium. This layer being the thickest at the gum margin (E), these papillæ are the longest and largest at this locality. The fibers of the tunica propria pass gradually into the submucous membrane (G), and from there into the periosteum and peridental membrane (M), so that it is difficult to determine the mucous capacity line of demarcation separating the different structures. The submucosa is composed of fibrous connective tissue of a much less compact variety. This structure is attached to the bones through the periosteum and peridental membrane. In this structure the glands, blood vessels, nerves, fat cells, etc., occur.

The larger blood vessels (K) are found in this structure. From these large blood vessels small capillaries extend to the tunica propria. It is here that inflammation commences in interstitial gingivitis when due to local irritation. Numerous veins accompany each artery, and lymphatics form a network around them. Small nerve filaments are also in this structure, which pass through the tunica propria and into each papilla in connection with the capillaries. The terminal nerve fibers come in contact with the muscular fibers, so that there is direct communication by blood vessel and nerve throughout the mucous membrane from the nose, stomach and lungs. The gum tissue is very thick and made up of fibrous tissue running in three or four directions, rendering it dense, tough and hard. The membrane thus differs from the same structure in other parts of the body. On account of these numerous fibers, this structure is bound tightly to the alveolar process. The gum tissue acts as a cushion and protection from irritation which may arise from hard substances being taken into the mouth. As this membrane passes and coalesces with the membrane of the lips and cheeks, it becomes much thinner and less dense. In the center of the tooth, the parallel fibers in the tunica propria are composed of flattened fasciculi of connective tissue. There are three sets of fibers—those which run vertically, those which radiate and are fan-shaped, and those which are horizontal.

X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 13.—LONGITUDINAL SECTION OF TOOTH AND GUM TISSUE. DOG.

D, Dentine. E, Epithelial Tissue. G, Submucous Membrane. K, Capillaries. M, Fibrous Tissue. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. RR, Space Pocket from Want of Union of the Epithelial Fold.

The mucous membrane, like the alveolar process and peridental membrane, is composed of very unstable tissue. It changes its structure, blood vessels and nerve system as often as the other structures. Its blood vessels and nerve system are continually renewing connective tissue, periosteum and peridental membrane.

A difference is noted in the structure of the papillary layer in man and the lower animals, such as the dog, the sheep and the calf. In man the gum tissue is not so thick, therefore the papillæ are broader and shorter, while in the lower animals the papillæ are narrow, long and more closely set together. Blood vessels and nerves are not so numerous and close together in man as in animals.

THE PERIOSTEUM AND PERIDENTAL MEMBRANE.

The periosteum is a fibrous tissue covering the outer surface of the alveolus. The peridental membrane is composed of similar structures covering the roots of the teeth and lining the inner wall of the alveolus. They are both derived from the mesoblastic layer. For this reason there can be very little difference in the character of the structure of each, except so far as function is concerned. The periosteum is made up of four different kinds of fibers. An outer layer of coarse, white fibrous tissue, an inner layer of fine, white fibrous tissue, elastic fibers, and penetrating fibers (fibers of Sharpey).

The fibers of the periosteum are coarser than those of the peridental membrane. The coarser fibers run parallel with the alveolar process (J) over the border and extend as far as the union of the epithelial layer (E) and the periosteum (H), Fig. 14. ("The dental ligament," Black.[2]) The finer fibers run in all directions and enter the alveolar process at every point. If a section of the alveolar process treated with acids or a section affected by halisteresis or osteomalacia be placed under the microscope, the fibers are seen to retain the original shape of the bone.

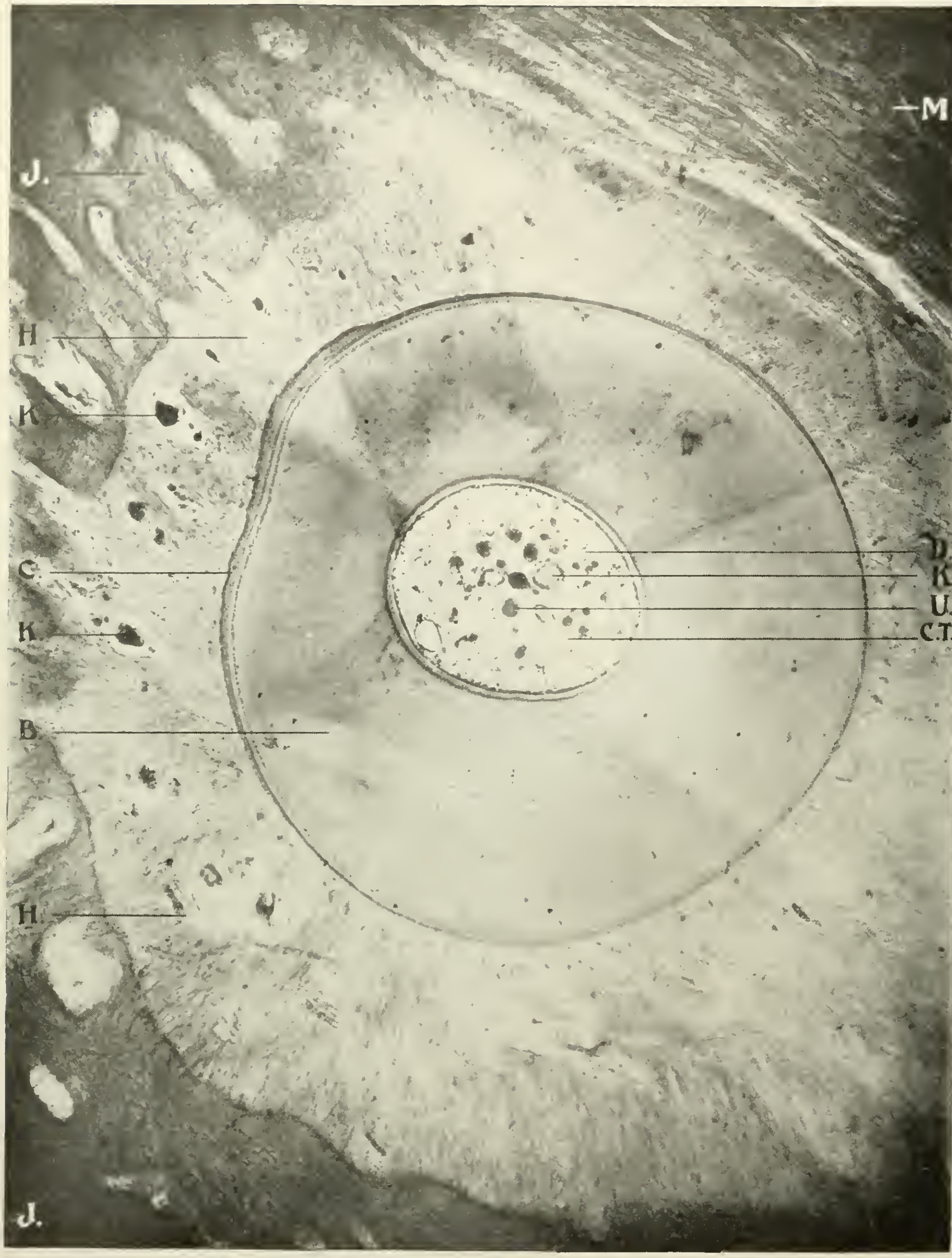
---

[2] American System of Dentistry, page 663.

X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 14.—LONGITUDINAL SECTION OF TOOTH, ALVEOLAR PROCESS, PERIDENTAL MEMBRANE AND PERIOSTEUM. NORMAL TISSUE. SHEEP.

B, Dentine. C, Cementum. E, Epithelial Tissue. G, Submucous Membrane. H, Periosteum. J, Alveolar Process. K, Capillaries. L, Haversian Canals. M, Fibrous Tissue. AA, Point of Union of Epithelial Tissue and Peridental Membrane.



X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

Fig. 15.—Cross Section of Tooth, Alveolar Process, Peridental Membrane and Periosteum. Normal Tissue. Dog.

B, Dentine. C, Cementum. D, Pulp. H, Periosteum. J, Alveolar Process. K, Capillaries. M, Fibrous Tissue. U, Nerve Tissue. CT, Connective Tissue.

The fibers of the periosteum, therefore, are continued throughout the process from the periosteum on the one side to the peridental membrane on the other. This is also illustrated in the mouths of persons, where (after wearing artificial dentures for a short time) heat produces absorption of the lime salts, leaving the fibrous tissues intact.

The periosteum is abundantly supplied with blood vessels which anastomose with each other and enter the alveolar process at the Haversian canals. The plexus of blood vessels is much larger proportionately in connection with the alveolar process than with other bones of the body, owing to its transitory nature.

The peridental membrane commences at the margin of the epithelium at the neck of the tooth AA, Fig 14, and is attached directly to the cementum. This membrane has various functions: First, it fills the space between these two structures, forming a cushion for the teeth to rest upon; second, like the alveolar process, it is present only when the teeth are present, and therefore develops with the alveolar process when the first teeth erupt, it is entirely lost when the temporary teeth are shed, is restored with the eruption of the second set, and when the permanent teeth are extracted it disappears with the alveolar process completely; third, it furnishes the nourishment for the teeth while they are in position in the jaw, and holds them in their sockets.

The fibrous tissue, in its earliest stages comprises nearly all or quite all of that portion of the jaw which eventually becomes the alveolar process. Calcification begins at the center of the jaws and gradually closes in upon the fibrous membrane until it becomes the thickness of a sheet of paper. In young persons the membrane is much thicker than in old age, since, as age advances, the osteoblasts on the one hand and the cementoblasts on the other send out new material and each wall closes in upon the membrane, which becomes very thin in old age and almost lost.

The fibers which compose this membrane extend in all directions; some crosswise penetrating the cementum, on the one hand, and the alveolar process on the other. In a general way,

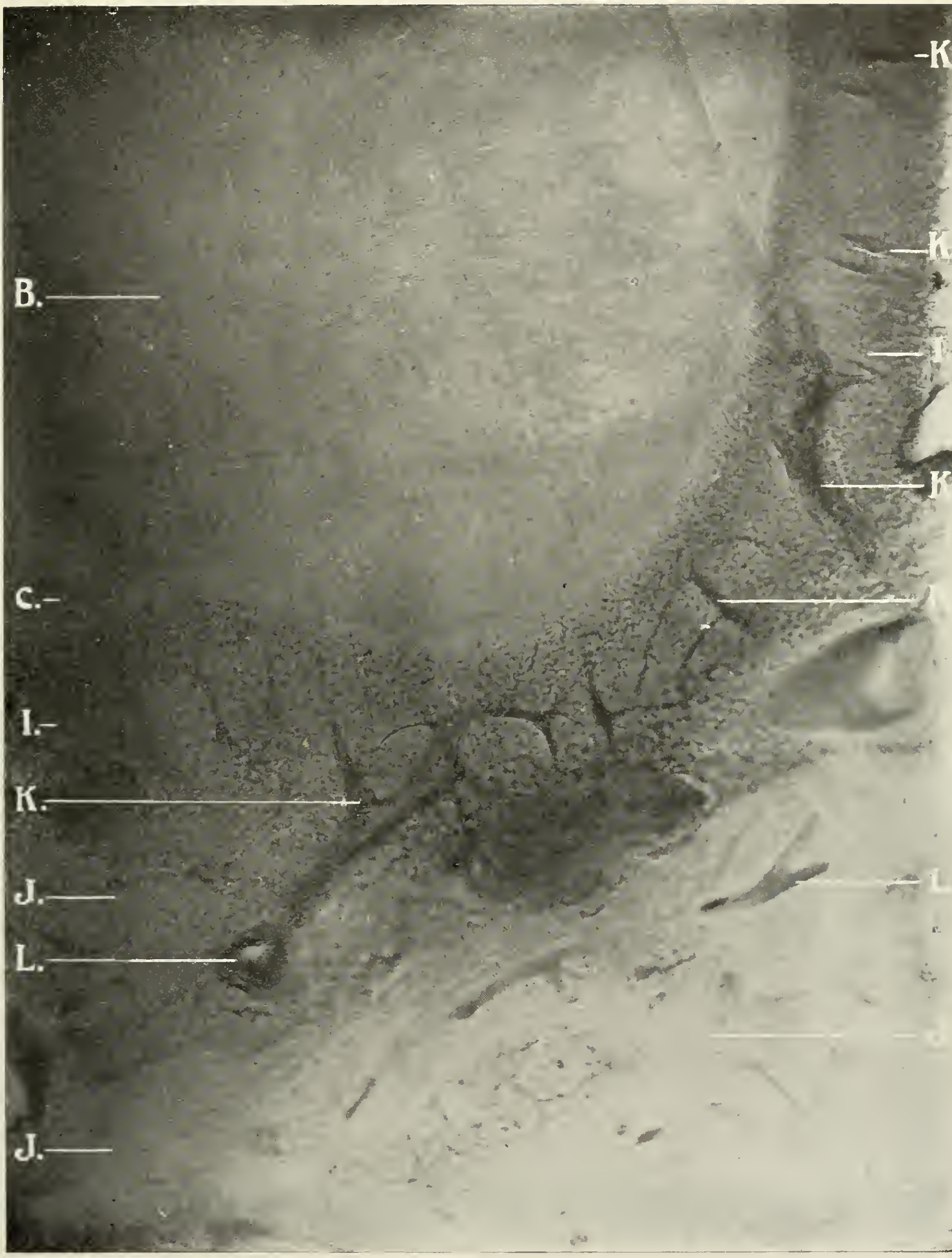
since the fibers extend through the alveolar wall, they are more closely adherent to the bone than to the cementum, and usually cling to the latter when the tooth is removed. It will be observed that these fibers do not enter the alveolar process uniformly as claimed by Gray[3] and Pierce[4], like tacks or nails driven regularly into a board (the "fibers of Sharpey" Fig. 14), but vary as to quantity in different localities. In some localities they penetrate in large quantities and almost surround a piece of alveolar process, while a few fibers penetrate but a short distance. In some places, they can be traced almost through the alveolar process. These fibers are much finer in man (Fig. 15) than in the lower animals (Fig. 14, dog). In connection with the fibers which pass into the alveolar process are numerous blood vessels. Others run diagonally, and still others lengthwise, all making up a tissue which holds the tooth in position in the jaw. The fibers enter the peridental membrane at all points of the process, from its margin to the apex of the roots. The elasticity of this membrane is so great that in correcting irregularities a tooth may be turned from one-fourth to one-half around without breaking the fibers. The elasticity is greatest in youth. As age advances, the membrane grows thinner and thinner until, late in life, there is almost a bony union between the tooth and the alveolar process, thus preventing stretching of the fibers. At the upper border, under the gum tissue, these fibers extend over the edge of the alveolar border and unite with the fibers of the periosteum on the outer border of the process, forming the interstitial tissue.

If absorption of the inorganic substance of the alveolar process occurs, the fibrous tissue retains the shape of the process. The same results when inflammation of the peridental membrane takes place at the gum margin or at the apex of the root of the tooth. What was once alveolar process is now peridental membrane or fibrous tissue.

Two kinds of structures are present in the alveolar process —a dense, compact, hard structure (composed of lime salts),

---

[3] Anatomy.
[4] American System of Dentistry, page 668.



X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 16.—CROSS SECTION OF TOOTH, ALVEOLAR PROCESS AND PERIDENTAL MEMBRANE. INJECTED BLOOD VESSELS. NORMAL. DOG.

B, Dentine. C, Cementum. I, Peridental Membrane. J, Alveolar Process. K, Capillaries. L, Haversian Canals.

X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

Fig. 17.—Cross Section of Tooth, Alveolar Process and Peridental Membrane. Injected Blood Vessels. Normal. Dog.

B, Dentine. C, Cementum. I, Peridental Membrane. J, Alveolar Process. K, Capillaries. L, Haversian Canals.

and a fibrous tissue; either alone will retain the shape of the tissue.

Blood vessels permeate this membrane throughout from the gum tissue at the neck of the tooth, through the alveolar walls to the end of the roots. They are most abundant in youth. Capillary blood vessels enter the Haversian canals through the process and into the cementum. Many of these blood vessels extend the entire length from the gum margin to the apex in straight lines and vice versa. In many of the illustrations, the blood vessels will be seen to follow the line of the alveolar process (Fig. 14). A great supply of blood vessels penetrate the membrane through the alveolar walls. These vessels unite and anastomose with the arteries which traverse lengthwise, forming a complicated plexus (Fig. 16). According to some writers the vascular supply of the peridental membrane is situated in the center of the structure. This has not been my experience. All of my slides, as well as those here presented, show the blood vessels to be situated nearest the alveolar process. It is quite natural that this should be so, since very little blood is required for the nourishment of the cementum, while the largest amount is required to supply the alveolar process. The system of blood vessels situated in the peridental membrane and showing their relation to the surrounding tissue is well shown in the injected specimen from healthy dogs (Figs. 16 and 17). Pus pockets and abscesses are hence more liable to form near and in the alveolar process than near the tooth structure. When infection takes place, the products of inflammation are carried through the blood vessel and the foci of round cell inflammation are located near or in the alveolar process where abscesses form. The vessels seen in the membrane anastomose very freely with those at the gum margin, showing the membrane to be well nourished in all its parts. Should one part become involved by disease the other parts are overnourished in consequence.

These blood vessels enter the alveolar walls with the fibrous tissue through the Haversian canals and these in turn permeate the entire bone. As age advances, however, the bone becomes more dense, and the Haversian canals become smaller and (under certain conditions) cease to exist. When disease takes

place, either at the gingivus or at the apex of the root, the supply of blood being thus cut off, the tissues receive sufficient nourishment through the alveolar wall. Since the structures are in a transitory state, being destroyed and repaired so frequently, it is evident why the blood supply is so rich.

CALCOSPHERITES.

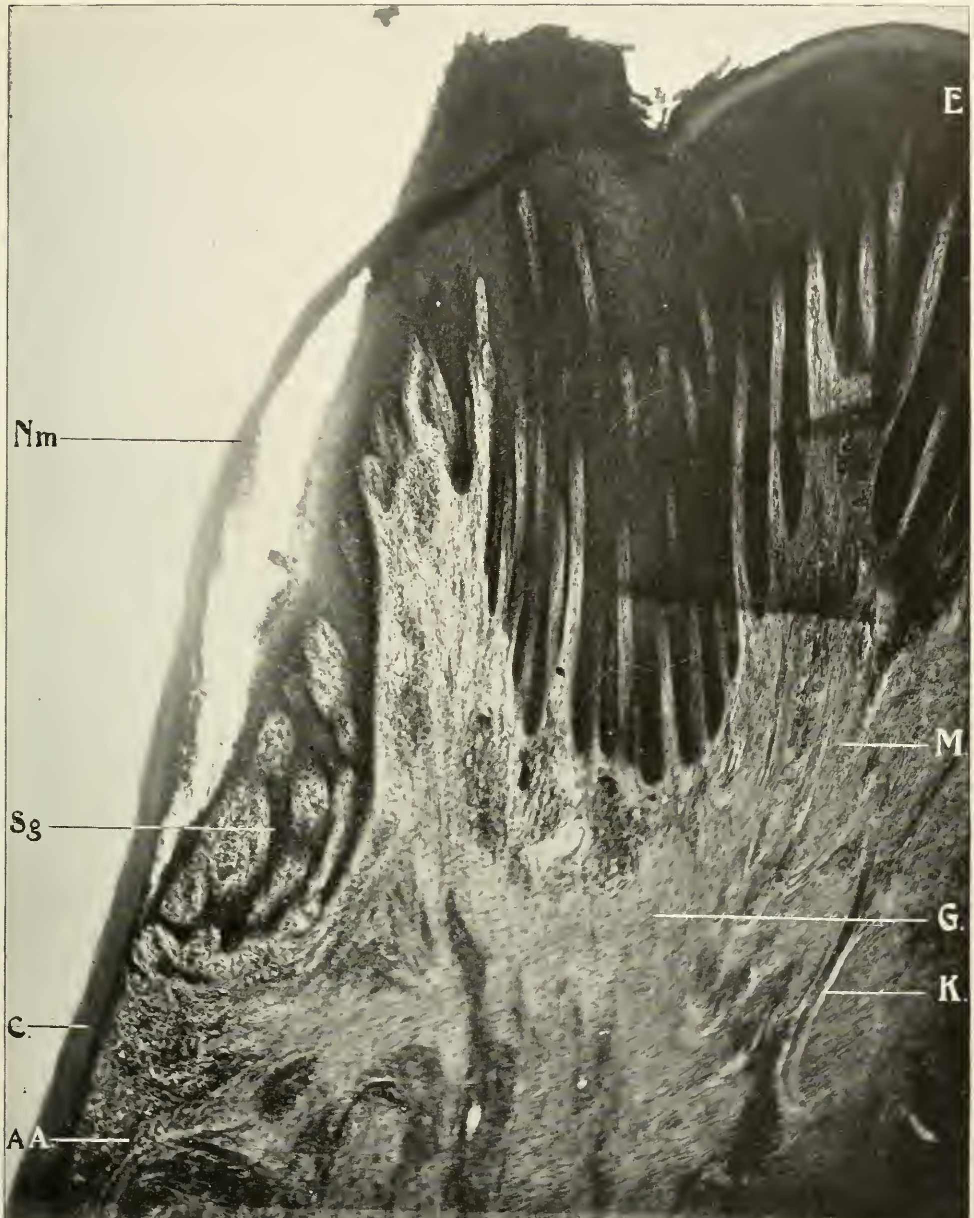
Small, hard bodies are frequently found in the peridental membrane. These are sometimes in the form of concentric rings of lime salts and are called calcospherites. They are not always round, but may be of any shape and vary as well in size. They bear the same relation to the peridental membrane that pulp stones do to the dental pulp. Black[5] says: "I have seen more of them about the roots of the molars than elsewhere, but have found them along the sides of the roots of the bicuspids." When irritation and inflammation take place in the peridental membrane, the cementoblasts build up cement substance, just as the osteoblasts do in the alveolar process and the odontoblasts do in the pulp chamber. Sometimes they are attached to the root of the tooth, producing a condition called exostosis or cementosis. They may remain unattached, floating in the fibrous tissue. These are very common in connection with interstitial gingivitis.

DO GLANDS EXIST IN THE MUCOUS AND PERIDENTAL MEMBRANES?

A somewhat widespread opinion locates special glands in the gingival tissues and the peridental membrane. This seems, to a certain extent, to be in part due to the lack of definite knowledge as to the etiology of interstitial gingivitis, and in part to the fact that certain constitutional conditions, such as mercurial and potassium iodid poisoning and scurvy, manifest themselves in the gum tissue in a way similarly to their action in the glandular structures of the body. Black[6] claims, for example: "That part of the gingival margin that lies in against the neck to the tooth is of a different structure from its other

---

[5] Periosteum and Peridental Membrane, page 94.
[6] American System of Dentistry, pages 955-956.



X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 18.—LONGITUDINAL SECTION OF GUM. NORMAL TISSUE. SHEEP.

C, Cementum. E, Epithelial Tissue. G, Submucous Membrane. K, Capillaries. M, Fibrous Tissue. AA, Point of Union of Epithelial Tissue and Peridental Membrane. Nm, Nasmyth's Membrane. Sg, So-called Glands of Serres.

parts. Here it is clothed with a very soft, round or polygonal gland-like epithelium that suggests the formation of a gland, but fails to assume the glandular structure, though it seems to have been regarded as such by Serres. This—which I shall call the gingival organ—emits a profusion of small rounded cells which are always found in the saliva (Salter) and are usually called mucous corpuscles. It is well known that certain glands have the power of the selection and excretion of certain poisons, and in this way of eliminating them from the system, and that if the substance be in large amount, hyperemia, or even inflammation, may result. It is also known that mercury and potassium iodid will produce inflammation of the free margins of the gums, and Salter has found these cells are in greater abundance under these circumstances; also that the cells taken from the gingival border and submitted to chemical tests after the person has taken potassium iodid are found to yield and are tinged with iodin."

Longitudinal sections of the tooth, alveolus and surrounding tissues, under the microscope, exhibit a very peculiar formation of the mucous membrane at its line of union with the peridental membrane at the neck of the tooth. Black[7] (in an article beautifully illustrated by Frederick Noyes) seems to identify these with the so-called glands of Serres or gingival glands (Fig. 18). He speaks of them as glands in various places throughout the article; for example, "But little can now be said of the function of the network of glands of the peridental membrane, beyond what is indicated by their form, location and histological characters. With the knowledge of their position and general character, clinical observation leads to the conclusion that they are readily disturbed by certain drugs, notably by mercury and iodin; and that they are often disturbed by substances poisonous to them floating in the blood streams. This is evidenced by the appearance of marginal gingivitis, with soreness of the peridental membrane. Such disturbances would not be likely to occur without the presence of some specialized or secretory tissue. . . . It seems to me very certain that the disease

[7] Dental Cosmos, February, 1899.

which I have described as phagedenic pericementitis has its seat in these glands.'' Black, however, does not seem quite certain of the validity of his position, since he further remarks, ''Though definitely lobulated, this body does not seem to possess the characters of a gland, and I should not suppose from an examination of its tissues that it had a glandular function. It encircles but a portion of the neck of the tooth, usually only the approximal portion, thinning away toward the buccal and lingual, so that in many of the lengthwise sections it may be very small, or does not appear at all.''

In many slides of sections from canine jaws and human,[8] the same peculiar arrangement of structure was observed, although not in so marked a degree. In the immature herbivora (calf and lamb) these peculiar formations of structure are well marked, albeit less so in the carnivora, and still less in man.

Were glands present in this locality it is logical to infer that they would become involved in mercurialism, plumbism and scurvy, and exhibit marked inflammation with broken-down structures in a given locality, as at the union of the gum tissue with the peridental membrane. Such a case is unknown.

The mucous membrane under the microscope appears at a point between the teeth (and faintly so at the inner and outer border as shown by Black) to double upon itself. When the tooth erupts, absorption of the gums occurs at the highest point. The gum tissue passing down to the neck of the tooth folds or crowds upon itself between the teeth with a peculiar curve downward, inward and then outward and upward. At the upper border, about midway from the gingival margin to the neck of the tooth, may be seen a space or pocket (never twice alike in appearance) where the edge of the gum tissue comes in contact with the original epithelium. Sometimes the space or pocket is closed up (Figs. 48, 49, 57). Again it remains open (Figs. 13, 37, 56, 59). Frequently this peculiar type of structure is absent, showing that the fold of gum tissue either has been absorbed in

---

[8] The material obtained for making slides from man, other than the scurvy cases, was obtained through the kindness of surgeons from jaws removed from hospital patients, as a result of disease; the surgeons placing them into alcohol or Müller's solution as soon as removed.

X 75. A. A. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 19. CROSS SECTION OF TOOTH AND PERIDENTAL MEMBRANE.

NORMAL TISSUE. SHEEP.

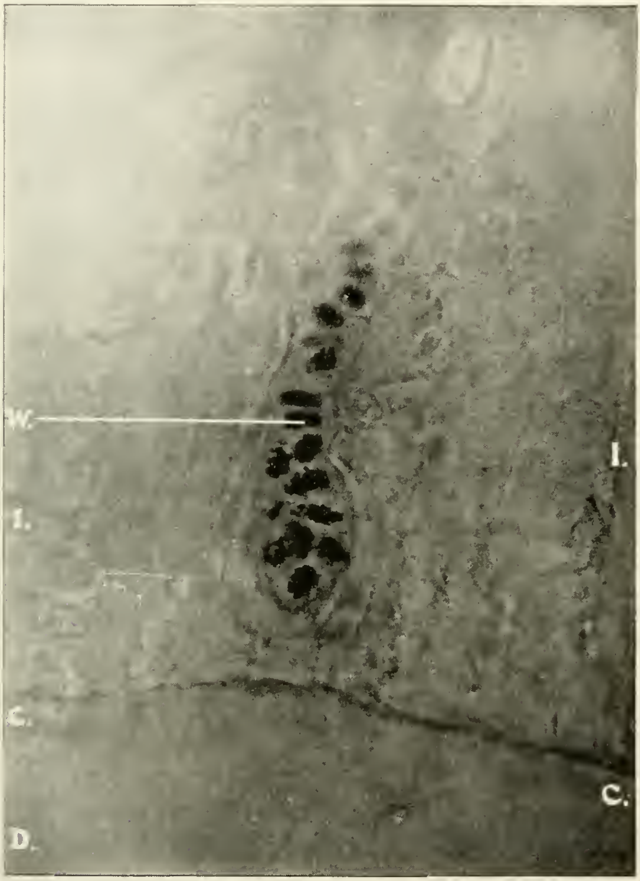
C, Cementum. D, Dentine. I, Peridental Membrane. W, Epithelial Débris.

X 300. No. 2 projection ocular. D. D. obj. Zeiss. Micro-photographs, reduced one-third.

FIG. 20.—CROSS SECTION OF TOOTH AND PERIDENTAL MEMBRANE. NORMAL TISSUE. SHEEP.

C, Cementum. D, Dentine. I, Peridental Membrane. W, Epithelial Débris.

the eruption of the tooth or did not form. This peculiar form encircles only a portion of the neck of the tooth (according to Black's examination of the structure in sheep). This in itself seems to offset the glandular theory, since gingivitis almost invariably starts on the lingual or palatine and labial surfaces



X 560. No. 2 projector ocular. One-twelfth obj. Zeiss.

Fig. 21.—Cross Section of Tooth and Peridental Membrane. Normal Tissue. Sheep.

C, Cementum. D, Dentine. I, Peridental Membrane. W, Epithelial Débris.

where this structure does not appear. In the slides of the scurvy case there does not appear the slightest evidence of anything resembling glandular structure. Hence it would seem safe to conclude that the glandular structure does not occur in this

locality. It is by no means impossible that in the peculiar epithelium in this locality, epithelial cells undergo changes which to some observers simulate glandular structure, but on histologic analysis are distinguishable from it, resembling in this the crypts of the head of the penis.

In cross sections of the peridental membrane, with a low power may be seen dark bodies arranged along the margin of the cementum in the peridental membrane (Fig. 19). They are more numerous, however, near the gingival border than at the root extremity. These bodies are more numerous and better defined in the sheep than in the calf, and more apparent in the canine jaw than in the human. Under higher power (Fig. 20) they may be distinctly demarcated as epithelial cells arranged in single rows of loops, again in double rows, again in rows of three and sometimes in round or oblong groups, with clusters of cells without shape or form. With a still higher magnifying power (Fig. 21) it will be seen that these masses of cells are polygonous, never prismatic. They hence are similar in shape to the epithelial cells situated above the columnar cells. They also resemble the cells which are situated inside of the epithelial lamina. In the larger amplification the nucleus can be readily observed.

Black[9] has attempted to demonstrate that glands exist in the structure and that the cells last mentioned are glands. Black lays down as a *sine qua non* of a gland that there should be an opening to the surface. He has made an attempt (Fig. 15) to demonstrate such an outlet, but this figure does not show clearly that the glands empty into the duct or have an exit at the surface. These bodies, however, not only fail (like the ductless glands) in this particular, but in more important characteristics of glands. They do not have (as Robin and Magitot remark) a columnar or prismatic cell wall. It is not difficult to understand how epithelial cells are scattered in different shapes and sizes throughout the peridental membrane. Epi-

---

[9] Dental Cosmos, February, 1899, pages 112-118.
[10] Dental Follicle, page 116.
[11] Embryology, pages 581-90.

thelial cells have the property of multiplying and developing in structures wherever located.[10]

If epithelial cells should migrate within the submucous membrane and fibrous tissue, proliferation will occur under certain circumstances. The tooth, according to Minot,[11] is a papilla which projects into the epidermis and, ossifying (calcifying) in a particular way, changes into ivory around the soft core or pulp; to the papilla the epidermis adds a layer of enamel. The tooth proper unites with a small plate of dermal bones at its base. By a modification in the jaws the epidermis first grows into the dermis and then the dermal tooth papilla is developed. The first indication of the development of tooth germs in mammals is a thickening of the epithelium covering the jaw. This thickening, which appears as a ridge during the sixth week of embryonic life, forms on the under side of the epithelium. This curving ridge expands into an outer portion (the outline of the groove between the lip and the gum) and an inner portion, the dental shelf which grows obliquely inward. The papillæ for the milk teeth are formed on the under side of the shelf, and it is thus possible for the shelf to continue growing toward the lingual side, so that the second set of germs is developed for the permanent teeth. The end of the shelf, toward the articulation of the jaws, is prolonged without retaining the direct connection with the epithelium and from this prolongation arise the enamel organs for the three permanent molars. Wherever a tooth-germ arises the dental shelf is locally enlarged, and the local enlargement constitutes an enamel organ which projects from the under side of the shelf. The portions of the shelf between the enamel organs gradually break up, forming first an irregular network, and later separate fragments[12] which may persist throughout life and lead to various pathological structures. While the permanent germs are forming, the shelf is solid between them, although it has assumed the reticulate structure between the germs of the milk teeth. In consequence of the reticular formation, the fully developed enamel organs

---

[12] Including the epithelial débris of Robin and Magitot.

have several bands or threads by which they are connected with the dental shelf proper.

After the shelf has developed somewhat, its line of connection with the epithelium of the gum becomes marked by a superficial groove, as may been seen in the human embryo of eight to ten weeks. This groove was formerly supposed to be the first trace of the dental shelf, but Rose's observations correct the supposition.

The second step in mammals is the formation of outgrowths (in man ten in each jaw) from the under side of the dental shelf; each outgrowth is the outline of an enamel organ for a milk tooth. The outgrowth is covered toward the mesoderm by a layer of the epidermis, while the core is filled with polygonal cells which resemble those of the middle part of the Malpighian layer of the skin. The outgrowths, after penetrating a short distance, expand at the lower ends, but remain each connected by a narrow neck with the overlying epidermis. The expanded end is the enamel germ proper; it very soon assumes a triangular outline, as seen in sections, owing to the flattening of its under side, and at the same time it moves somewhat toward the lips. Meanwhile the shelf continues growing on the lingual side of each ingrowth to produce the enamel organs destined for the second or permanent teeth.

At this stage it is noticed that the mesenchyma under the flattened end of the enamel organ has become more dense, to form the outline of the dental papilla, and is beginning to develop fibrillæ around both the enamel germ and the papillary outline. The fibrillar envelope is the future dental follicle.

The third step is a final differentiation of the enamel organ and the accompanying shaping of the papilla. The enamel organ continues growing and becomes concave on its under side so that the mesoderm underneath acquires the shape of the papilla. It is now that the form of the tooth is determined by the form assumed by the papilla, which in its turn is probably determined by the growth of the enamel organ.

The follicle is merely an envelope of connective tissue in which can be distinguished an outer dense and inner looser layer; in the latter the cells are more distinct and the fibrillæ

are less numerous than in the former. A rich network of capillary vessels is developed in the follicle and appears in part as a series of villous-like growth into the enamel organ. The follicle develops first over the lower part of the papilla, then over the enamel organ, the neck of which aborts and the follicle closes over, completely separating the enamel organ from its parent epidermis. The enamel organ changes greatly in appear-

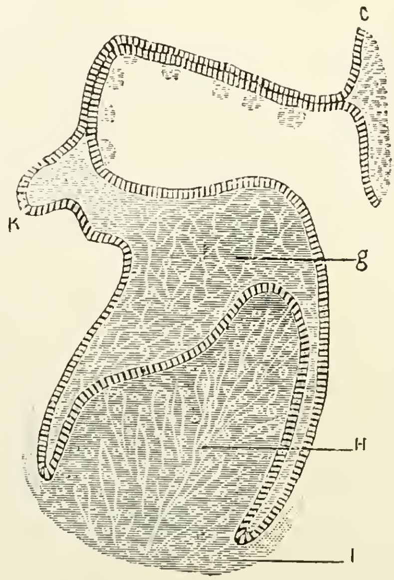


Fig. 22.—Section through the Incisive Portion of the Lower Jaw of an Ovine Embryo, Measuring 82 Millim. (3 1/3 Inches) in Length. Magnified 260 Diam., after Drs. Ch. Legros and E. Magitot.

D, Oral Epithelium. C, Lowest Layer of Cells in the Stratum Malpighii. F, Epithelial Cord. K, Bourgeon of the Secondary Cord. I, Follicular Wall. H, Dental Bulb.

ance. The layer of cylinder cells is well preserved over the concave surface, but only where the epithelium is in contact with the dental papilla. In the neck the cells become irregular in form. Over the convex surface the cells become lower and cuboidal. They ultimately atrophy and flatten out. The cells in the center of the enamel organs undergo a peculiar metamorphosis. They remain united together by a few thread-like processes.

It is obvious from these changes in the embryo how what Robin calls the epithelial debris is derived from the epithelial

cord, the follicular wall and the round bodies of lamina epithelium debris. According to Ch. Robin and Magitot,[13] who were the first to describe these bodies, "The phenomena of budding commences, namely, when the epithelial cord has finished its course, having conducted the primary enamel organ to that point whence its subsequent evolution will be effected and soon after the formation of the secondary follicle, immediately after the rupture of the cord of the primitive follicle." Robin leans to the opinion that these bodies disappear soon after they are formed. "The time of their disappearance varies, they remark, in different species of animals. In the human embryo the

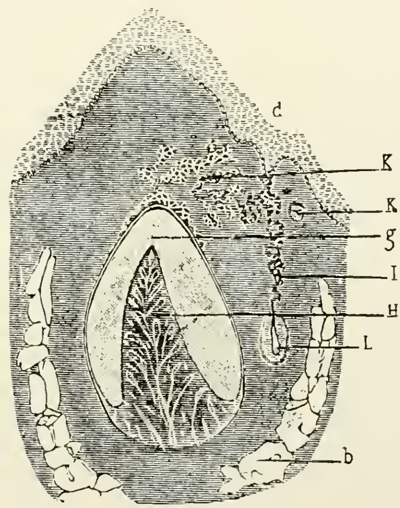


FIG. 23.—VERTICAL TRANSVERSE SECTION THROUGH THE INCISIVE REGION OF THE LOWER JAW OF HUMAN FOETUS MEASURING 38 CENTIMETRES (15½ INCHES), MAGNIFIED 80 DIAM., AFTER DRS. CH. LEGROS AND E. MAGITOT.

b, Bony Formation. d, Oral Epithelium. g, Enamel Organs. H, Dental Bulb. I, Cord of the Permanent Follicle. K, Débris on the Follicular Wall of the Primitive Follicle and from its Cord. K, Epithelial Globule. L, Enamel Organ of the Permanent Tooth.

remains of the cord of the primitive follicles may be found, even after the formation of the follicles of the permanent teeth, and it is probably during the process of eruption that these buddings become atrophied; in the canine embryo the facts are nearly the same; in the bovine and ovine embryos (calf and lamb) it has seemed to us that these proliferations disappear at

---

[13] See their Memoir on the Genesis and Development of the Dental Follicle in Jour. de Physiologie de Brown-Sequard, 1860.

a correspondingly earlier stage; and we think it safe to say that, as a general rule, the complete absorption occurs toward the period of eruption.''

The embryology of the dental shelf, which has been summarized by Minot from Waldeyer, Kolliker, Von Ebner[14] and O. Hertwig, indicates the source of the structures which have been mistaken by Black for the limiting walls of glands.

After the epithelial cords of the temporary and permanent sets of teeth have been demarcated from their follicles, the proc-

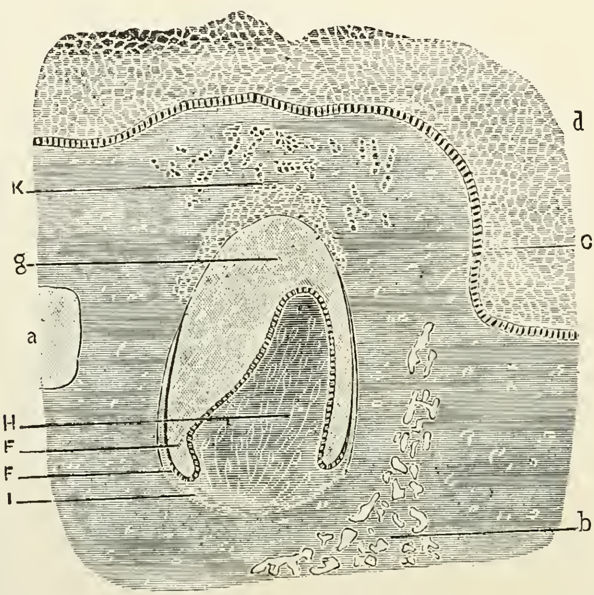


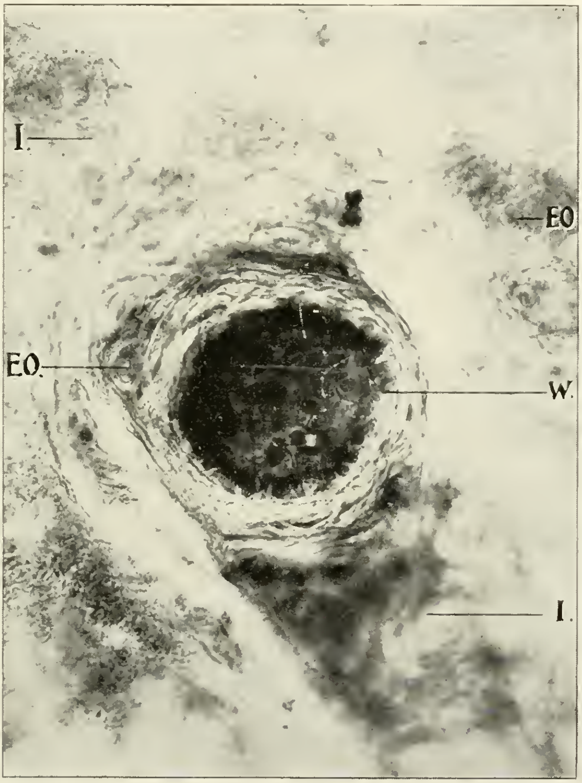
FIG. 24.—FROM THE LOWER JAW OF AN OVINE EMBRYO, MAGNIFIED 80 DIAMETERS, SHOWING THE COMPLETED DENTAL FOLLICLE AND THE SURROUNDING TISSUES, AFTER DRS. CH. LEGROS AND E. MAGITOT.

a, Meckel's Cartilage. b, Traces of Ossification. c, Lowest Layer of Epithelial Cells. d, Oral Epithelium. F, Ameloblastic Layer. F, (Lower) External Layer of the Enamel Organ—a continuation of the Layer of Ameloblasts. g, Stellate reticulum of the Enamel Organ. H, Bulb. I, Follicular Wall. K, Buddings from the Cord.

ess of cell building proceeds like the process of cord building. These buds, according to Charles Robin and Magitot,[15] are given off at the upper border of the follicle and below the epithelium of the gum. ''In fact, as soon as the epithelial lamina loses its connection with the follicle, by the rupture of the cord, the epi-

[14] Handbuch der Zahnheilkunde, 1890, pages 209-262.
[15] Loc. cit., 1860.

thelial cells composing it become greatly increased in number at the severed point. The multiplication of cell-elements results in the formation of irregular buddings, which wander in different directions into the deeper portions of the embryonal tissue. These buddings vary greatly in form; sometimes they are simple cylinders, retaining their connection with the primitive lamina by pedicles of various lengths, and sometimes this slight



X 50. One-half-in. obj. No, Oc.

FIG. 25.—CROSS SECTION OF EPITHELIAL CORD. MAN.

I, Peridental Membrane. W, Epithelial Débris or Cord. EO, Endarteritis Obliterans.

connection is absorbed, thus isolating an epithelial mass."

This budding occurs at different points along the cord (Fig. 22) at the end and upon the outer surface of the follicular wall (Figs. 23 and 24) at the point where the cord is severed from the enamel organ. These gradually diminish as they descend upon its sides. Doubtless the epithelial cord remains in the periosteal

and submucous tissue throughout life. Fig. 25 represents evident sections of epithelial cord in a man sixty-eight years of age, and Fig. 26 in a dog eight years. In the photographs of the scurvy cases and of dogs will be seen evidences of the persistence of epithelial debris late in life. The position already cited from Robin and Magitot as to its early disappearance would hence appear to be too strongly taken.

Robin and Magitot claim that this budding process occurs at or about the time of the rupture of the cord. Up to this period ossification has not taken place, but then deposits of bone appear in the fibrous tissue of the middle and outer surfaces. The bone deposit gradually takes the form of the jaw, filling in and encroaching upon the fibrous tissue, forming a bony wall on the one hand, and the crown and root of the tooth on the other. When the tooth is ready to erupt, the crown pushes the soft tissue laterally, while the root develops, forms a defined wall with the peridental membrane between them. The epithelial debris (to use their term) which before was scattered over the entire surface of the dental follicle, is now crowded into the very narrow space of the peridental membrane, and owing to the position of the debris at the upper part of the follicle, it would be natural to find most of it at the peridental membrane.

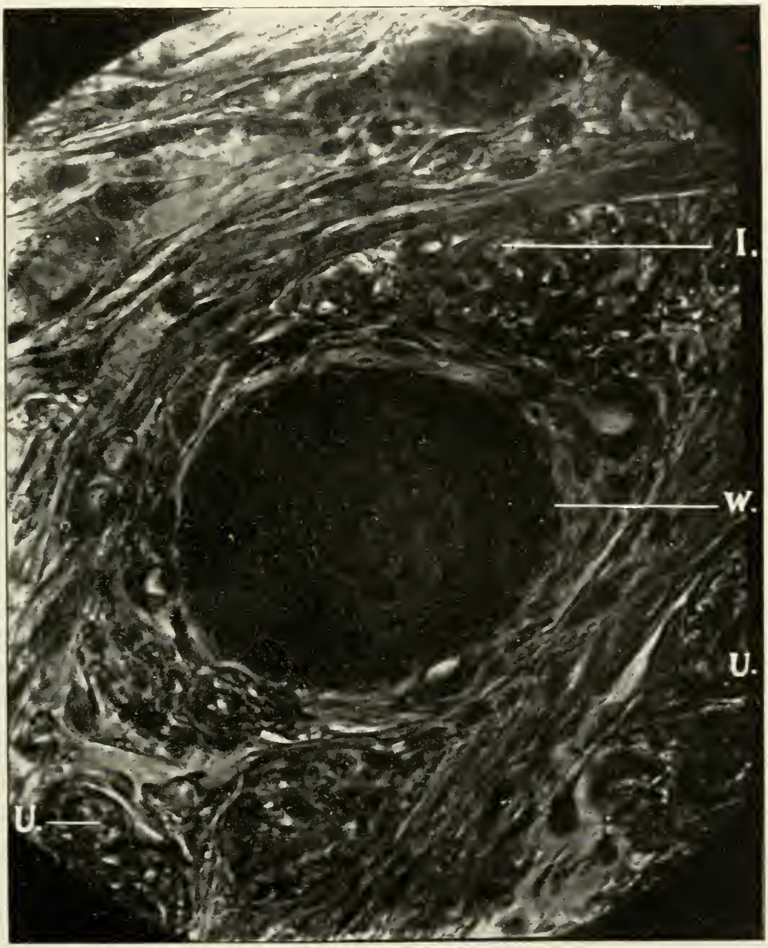
BONE BUILDING AND ABSORPTION.

Development of the alveolar process is relegated to a series of cells situated in the fibers of the peridental membrane or fibrous tissue, and close to the margin of the bone tissue, and throughout the Haversian canals. These cells are called the osteoblasts. They perform the function of building up the bone tissue. Even after the alveolar process has developed its normal shape, so unstable is the nervous system which presides over these cells at this locality, that at the slightest provocation, either local or constitutional, they will continue their process of construction. Hence, the frequency of hypertrophy of the process, and in disease the calcification of the peridental membrane.

On the other side of the membrane, next the root surfaces, may be seen other cells which build up and destroy the cemen-

tum; these are called cementoblasts and cementoclasts. These are of little importance in this connection with the study of this disease, although they are frequently present and at work when inflammation of the membrane occurs.

There is, however, another class of cells found in the peri-



X 560. No. 2 projection ocular. One-twelfth obj. Zeiss.
FIG. 26.—CROSS SECTION EPITHELIAL CORD. DOG.
I, Peridental Membrane. W, Epithelial Débris or Cord. U, Nerve Tissue.

dental membrane of the utmost importance in this connection, the osteoclasts, located in the fibers, and in close proximity to the alveolar wall, and around the inner border of the Haversian canals. The function of these cells is to tear down irregular

bone and tooth structure due to unstable nervous tissue, and from the slightest irritation.

The gums, mucous membrane, alveolar process and peridental membrane, owing to their transient nature, are influenced by the slightest irritation. This influence is the result of both constitutional and local causes. It consists of an irritation in the peripheral nerves which sets the osteoblasts and osteoclasts at work to build up or tear down the alveolar process. This influence may be only sufficient to stimulate these cells to action without inflammation. This is noticed in the advance toward old age, in long, lingering debility, in the development of bone, especially the tearing down and the building up of the inferior maxillary backward. It may be noted in mild or intense inflammation of the peridental membrane, due to more acute forms of disease, to scurvy, mercurial, lead and iodide poisoning, or to local irritation. So sensitive are these structures that in neurotics and degenerates the slightest irritation produced in the physiologic development of the permanent teeth is sufficient to start the osteoblasts to building up bone structure, thus producing that pathologic condition called hypertrophy of the alveolar process; one of the most marked evidences of an unstable nervous system.

The breaking down of the tissues by the osteoclasts may be induced by as slight a cause. The alveolar process being so thin about the teeth, destruction of the entire walls is accomplished without difficulty, and in a very short time, thus loosening the teeth, which eventually drop out.

According to Kaufmann [16] the following processes take part in the absorption of bone: (a) Lacunar Absorption, (b) Formation of Perforating Canals, (c) Disappearance after Prior Absorption of Lime (Halisteresis) (Bony Waste) and Osteomalacia.

"By far the commonest form of bone destruction is by lacunar absorption. This process occurs not only under physiologic conditions, but is extraordinarily frequent in pathologic states, e. g., in the various types of atrophy. They form on the

[16] Pathologische Anatomie.

smooth, superficial surfaces of the bone deep grooves (so-called Howship's lacunæ) in which lie smaller or greater polynuclear cells (osteoclasts, Kolliker) which evidently blend together (Fig. 27). There are no alterations of the bone substance that would indicate a primary line of absorption (Plummer). The confluence of these lacunæ form larger cavities.

"The second form of bone absorption, which is occasionally met under physiologic conditions, is by means of perforating canals (so-called Volkmann canals). Under physiologic conditions canals occur in varying numbers in the lamellæ (general

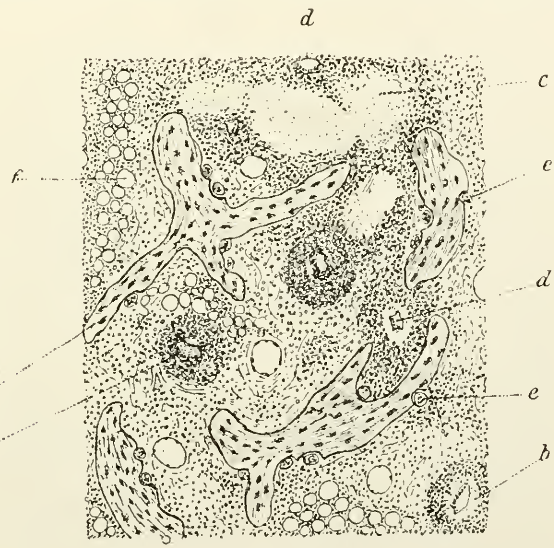


Fig. 27.

a, Bone Trabeculæ. *b*, Tubercle with Granulation Tissue. *c*, Broken-down Tissue. *d*, Blood Vessel. *e*, Osteoclasts. *f*, Fat Cells. (Kaufmann.)

lamellæ) which contain vessels (perforating vessels). These are often associated with the Haversian canals and gradually pass into them, but unlike them, are surrounded with circular lamellæ. Under pathologic conditions the conception of these perforating canals is somewhat widened. On the one hand Volkmann's canals are spoken of when reference is made to the vessels or vascular connective tissue penetrating from one medullary space in the spongy substance, or from one Haversian canal in the compact substance, to another, in such a way that a passage is made from one part of the bone to the other; Volk-

mann's canals also include irregular ampula-formed dilations or cavities (Fig. 28). By confluence of these are produced cavities or irregularly outlined canals penetrating the bone substance. These, if they empty into the medullary space, become filled with cells.

FIG. 28.

a, Large Spaces Resulting from Absorption of the Trabeculæ. *b*, Decalcified Bone. *c* and *d*, Decalcified Bone and Atrophied Trabeculæ. *e*, Haversian Canals. (Kaufmann.)

"Under much rarer conditions, especially in senile marasmic osteomalacia and also in that occurring in pregnancy, bone absorption takes place after a prior abstraction of lime (halisteresis) and the remaining substance (bone cartilage) is then fur-

ther dissolved, passing through a temporary fibroid stage. This destruction of the decalcified and interfibrillæ decomposed bone is produced as a rule without osteoclasts. The decalcified border zones of the trabeculæ appear with simple carmine (coloring) or by double stains."

Viewing the alveolar process, including the gums, peridental membrane and periosteum in man's ontogeny as a whole, it will be seen that the changes which are going on in apparently normal individuals make it an exceedingly transitory structure.

In connection with what has already been said in regard to the transitory nature of the alveolar process, there is another factor to be considered which makes it a doubly transitory structure.

In man's phylogeny, in some of the lower vertebrates [17] there is a continuous succession of teeth throughout life. In man's ontogeny, it would be strange if he did not retain still further evidences of phylogenetic peculiarities in tooth development in relation to the alveolar process.

Man and some of the lower vertebrates have only two sets of teeth. When the first set comes into place, the alveolar process builds itself up about the roots to hold the teeth in place. When these are to be lost, a low form of inflammation sets up absorption of the bone and the teeth are lost. When the second set erupt new bone is developed about the roots to hold the second set in place. Should man live long enough the second set would drop out even though he possessed a normal healthy body. This process is atavistic. With all these changes going on in man's ontogeny, we again have an exceedingly transitory structure. With the phylogenetic and ontogenetic changes, man has a doubly transitory structure in the alveolar process. I have called this process of bone absorption, osteomalacia or juvenile or senile absorption according to the age of the patient.[18]

Transitory structures are more easily involved in disease than other structures. How much more quickly then will a structure which is doubly transitory become involved in disease?

---

[17] Talbot. Developmental Pathology: A Study in Degenerative Evolution.

[18] Talbot. Pathogeny of Osteomalacia or Senile Atrophy. The Dental Digest, August, 1903.

Mammals (upon whom researches have been conducted by the author) demonstrate they are phylogenetically subject to this disease and perhaps lower vertebrates, including the reptiles under similar circumstances or environment.

The Alveolar Process as an End Organ.[19]—I have called the alveolar process an end organ. My reason for doing this is that the tooth, so far as the process and its diseases are concerned, is a foreign body.[20] The arteries, vessels of Von Ebner and especially the nerves pass through the bony process, in a wavy manner and stop at the root of the tooth.

There are other end organs in the body, chief of which are the kidneys, the eye and the brain. Physicians claim, and rightly, that because these are end organs they are more easily involved in disease and are often the determining factors of kidney lesions. Alfred C. Croftan says, "It is not surprising to find that particularly those organs that are supplied by end arteries are chiefly involved, for in them vascular disturbances must first produce nutritional derangement. Chief among the organs supplied by end arteries are, precisely, the kidneys, the retina and the brain, and I think this explains the frequent involvement of the kidneys, eyes and brain in Bright's disease. The fact that the retina and the brain are often found injured before the kidneys, that cases of Bright's disease run their fatal course occasionally with practically no renal changes, but with serious apoplectiform brain lesions and retinitis, bears out this conception and constitutes a valid argument against the common belief that the nephritis is the primary event and the determining phenomenon of the disease."

A marked difference exists between the kidney, eye and brain as end organs and the alveolar process as an end organ. This difference is the important point in the study of interstitial gingivitis. End arteries running into the kidney, eye and brain, owing to the soft nature of these tissues, are given a chance to expand and recover, permitting, in a measure, the blood to flow more easily, thus prolonging the tendency to disease, or allow-

---

[19] Endarteritis Obliterans. The Dental Digest, October, 1903.

[20] Interstitial Gingivitis or So-called Pyorrhœa Alveolaris. The Dental Summary, 1903.

ing the tissues, under favorable conditions to recover. On the other hand, blood vessels extending throughout the alveolar process in a tortuous manner cannot expand, and as a result, blood charged with toxins and subject to cardio-vascular changes immediately sets up irritation and inflammation which results in dilatation, bone absorption and arterial degeneration. These changes, therefore, will occur much earlier in the alveolar process than in other end organs.

The transitory nature of the alveolar process, especially as an end organ, makes it exceedingly sensitive to systemic changes and disease. The sensitiveness of this structure to autotoxic states is easily demonstrated as people advance in years. At the fifth period of stress (about forty-five and beyond) the excretory organs weaken. The toxic elements of the body are not carried off as freely as formerly. These circulate in the blood and accumulate in the alveolar process, setting up irritation and inflammation. Absorption of the alveolar process gradually takes place. People enjoying apparently good health will, as they advance in years, note the absorption of the alveolar process and the exposure of the roots of the teeth. How much more readily will absorption take place when the function of any one of the eliminating organs be involved, such as constipation, asthma, skin affections or kidney lesions.

In addition to the alveolar process being a doubly transitory structure it is also an end organ, of a bony nature and the most sensitive structure of the human organism, hence any constitutional disturbance due to disease, drug poisoning or autointoxication would more quickly affect it. Sudden changes in temperature from heat to cold and vice versa, where the organism is unable to adjust itself readily, also leave their mark upon the very susceptible tissue.

The study of interstitial gingivitis and its treatment must be based upon the phylogeny, ontogeny and peculiar anatomy of the structures involved. There are no other structures in the human body associated like the jaws, alveolar process and teeth. The pathology, therefore, is unique in itself.

Transitory structures in the body as well as end organs are known to be very susceptible to disease and are, as a rule, the

first to be involved. The alveolar process, being classed as a doubly transitory structure as well as an end organ consisting of bone, is one of the first, if not the first, structure to register systemic changes.

CHAPTER VII.

INORGANIC SALTS AND INTERSTITIAL GINGIVITIS.

The foods which enable the body to repair its waste, to build up new tissue and to supply the energy, are divisible into four classes: the inorganic substances, the fats or hydrocarbons, and the starches and sugars, or carbohydrates, and the proteid compounds. These divisions are, however, relative, since the proteids may contain both hydrocarbons and carbohydrates. The inorganic substances, such as water, phosphates, chlorides, carbonates, sulphates, etc., enter the body, as a rule, under their own form, either alone or in combination with other classes. They are not oxidized or split up within the system to enter into the chemical formations of other compounds, but are united mechanically with the proteid group. These bodies act, as a rule, in a purely mechanical manner. After having served their purpose, they pass out of the system with the excretions, comparatively unchanged in their composition. They are the only member of the group of foods which are of a special interest in the present research. The inorganic salts have not received the attention from physiologic chemists that their importance demands. They are, as a rule, found in greater or lesser quantities in all foods taken into the body. They do not serve as a source of energy, but as the other foods are needed for the development of the tissues, so the inorganic salts are needed for the building of bone tissue and the repair of waste. This is accomplished by the soluble salts in the blood. Human blood has the following composition:

ANALYSIS OF HUMAN BLOOD (C. SCHMIDT). HOWELL'S PHYSIOLOGY.

	Man. 25 Years.	Woman. 30 Years.
Water	788.71	824.55
Solids	211.29	175.45
Proteids and Extractives	191.78	157.93
Fibrin	3.93	1.91
Hæmatin (and iron)	7.70	6.99
Salts	7.88	8.62

INORGANIC SALTS OF HUMAN BLOOD, 1,000 PARTS (C. SCHMIDT).

Blood Corpuscles.		Blood Plasma.	
Cl	1.75	Cl	3.536
K_{20}	3.091	K_{20}	0.314
NA_{20}	0.470	NA_{20}	3.410
SO_3	0.061	SO_3	0.129
P_{205}	1.355	P_{205}	0.145
CaO		CaO	
M_{90}		M_{90}	

These acids and bases exist, of course, in the plasma and the corpuscles as salts. It is not possible to determine exactly how they are combined as salts, but Schmidt suggests the following combination:

PROBABLE SALTS IN THE CORPUSCLE.

Potassium Sulphate	0.132
Potassium Chloride	3.679
Potassium Phosphate	2.343
Sodium Phospate	0.633
Sodium Carbonate	0.341
Calcium Phosphate	0.094
Magnesium Phosphate	0.060

PROBABLE SALTS IN THE PLASMA.

Potassium Sulphate	0.281
Potassium Chloride	0.359
Sodium Chloride	5.546
Sodium Phosphate	0.271
Sodium Carbonate	1.532
Calcium Phosphate	0.298
Magnesium Phosphate	0.218

It will be seen that the corpuscle contains an excess of potassium salts, and the plasma contains an excess of sodium salts. All parts of the blood contain salts, however.

Throughout the entire body, there is a rich supply of blood vessels penetrating every tissue. The plasma of the blood passing by exosmosis through the walls of the capillaries is thus brought in immediate contact with the tissues to which it brings nourishment and oxygen of the blood, and from which it removes the waste products of metabolism. Other usable products or lymph are collected in small capillary spaces, which in

turn open into definite lymphatic vessels. These vessels unite into larger and larger ones, which eventually pour this usable waste product into the great thoracic or left lymphatic ducts, and a second smaller right lymphatic duct. These in turn empty into blood vessels, each upon its own side. The lymph contains essentially the same constituents as the blood plasma, and the salts are found in the same proportion as in it. They are then eliminated through the sweat glands, tonsils, mucous glands, kidneys, large intestines and salivary glands.

The composition of the deposits in the various parts of the body vary according to the locality and the character of the excreta eliminated in connection with them.

The chemical composition of the human bile, according to Jacobson,[1] is as follows:

Water	977.40
Sodium Glycocholate	9.94
Cholesterin	0.54
Free Fat	0.10
Sodium palmitate and sterrate	1.26
Lecitine	0.04
Other organic matter	2.26
Sodium chloride	5.45
Potassium chloride	0.28
Sodium phosphate	1.33
Lime phosphate	0.37
Sodium carbonate	0.93

Of this analysis the solid ingredients constitute 22.5 parts per thousand, of which two-thirds are organic and one-third inorganic. The inorganic salts of the bile are in most cases returned to the blood, where they are redistributed to the tissues. Occasionally, however, gall stones occur, which are composed of (analysis H. D. Geddings [2]):

Moisture	3.32
Biliary matter	32.182
Cholesterin	54.952
Matter soluble in ether	7.77
Iron	traces
Phosphoric acid	traces
Lime	traces
Magnesium	traces

[1] American System of Dentistry.

[2] Transactions South Carolina Medical Association, 1880.

Secretions of the pancreatic juice (dog) by C. Schmidt are as follows:

Water	900.76
Solids	99.24
Organic substances	90.44
Ash	8.80
Sodium carbonate	0.58
Sodium chloride	7.35
Calcium magnesium and sodium phosphate	0.53

The composition of the normal human pancreatic juice has not been determined completely owing to the difficulty of obtaining the secretion. According to Zawadsky the composition of the secretion of a young woman was as follows:

Water	in 1,000 parts	864.05
Organic substance	in 1,000 parts	132.51
Proteids	in 1,000 parts	92.05
Salts	in 1,000 parts	3.44

INORGANIC SALTS IN THE SWEAT.

"Of the inorganic salts, NaCl is by far the most abundant; it occurs in quantities varying from 2 to 3.5 parts per thousand. The elements of the sweat which are of importance from an excretory standpoint are water, inorganic salts and urea or related nitrogenous compounds."

Inorganic salts from the fæces are made up of the salts of sodium, potassium, calcium, magnesium and iron. According to Enderlin[3] the following represent the composition of the material matter in the fæces:

SALTS SOLUBLE IN WATER.

Sodium phosphate	2.63
Sodium chloride and sulphate	1.37

SALTS INSOLUBLE IN WATER.

Earth phosphate	80.37
Ferric phosphate	2.09
Calcium sulphate	4.53
Silicic acid	7.94

Like other constituents of the lymph, the salts vary considerably in proportion, according as the fluid is more or less rich in

[3] Gamgee, Physiological Chemistry of the Animal Body.
[4] American Text-Book of Physiology.

water. The salts are much more abundant than the organic solids.

Inorganic salts in the urine consist, according to Howell,[4] chiefly of chlorides, phosphates and sulphates of the alkalies and the alkaline earths. As a rule they arise partly from the salts ingested with the food, which salts are eliminated from the blood by the kidney in the water secretion, and in part they are formed in the destructive metabolism which takes place in the body, particularly that involving the proteids. Sodium chloride occurs in the largest quantities (about 15 grams per day), of which the greater part is derived directly from the salt taken in the food. The phosphates occur in combination with Ca and Mg, but chiefly as acid phosphates, of Na or K. The acid reaction of the urine is caused by these latter. The phosphates are produced in part from destruction of phosphorous-containing tissues in the body, but chiefly proceed from phosphates in the food. Following are the average quantities in grams of the chief substances normally excreted in the urine in six hours:[5]

Water	1440	—	1500
Solids	57	—	68
Organic:			
Urea	28	—	68
Uric acid	7		
Hippuric acid	3	—	2
Kreatinin	1.7	—	2.1
Inorganic:			
Sodium chloride	15	—	20
Phosphoric acid	2.5	—	3
Sulphuric acid	2	—	2.5
Sodium	5	—	7
Magnesium	.04		
Potassium	3	—	4
Calcium	.03		

Urinary calculi (classified according to their principal ingredients), are divided into:

1. Uric stone, composed of uric acid and acid urates.

2. Oxalic stone, composed of lime oxalate.

3. Phosphoric stone, which are composed of magnesium phosphate and carbonate with urate of ammonia.

[5] Landolt, Physiology.

Each one of these compounds is nearly in a pure state. A stone may be composed entirely of one salt or it may be composed of two, three or four, each compound forming separate consecutive layers through the stone. One examination made by Howship Dickinson [6] showed eighty-nine per cent lime carbonate and the rest lime oxalate and phosphate of lime.

The deposits upon the teeth are derived partly from the salts ingested with foods, which salts are eliminated from the blood in water secretion, and in part they are found in the waste of tissue which takes place in the body.

The saliva, according to Schmidt, is made up of the following:

Water	991.45
Organic material	2.89
Inorganic:	
Calcic chloride	4.50
Sodium chloride	....
Calcic phosphate	1.16
Magnesium	....
	1,000.00

This material floating in the saliva, together with the epithelial scales and other extraneous matters, contribute to form what is known as tartar. This material collects upon the teeth, and according to examinations by Stevenson consists of:

	Soft tartar on molars.	Hard tartar on lower incisors.
Water and organic matter	21.48	17.51
Magnesium phosphate	1.31	1.31
Calcium phosphate with a litte carbonate and trace of flourine	77.21	81.18
	100.00	100.00

Another analysis made by Schehoetskey resulted thus:

Water and organic matter	22.07
Magnesium phosphate	1.07
Calcium phosphate	67.18
Calcium carbonate	8.13
Calcium flouride	1.55
	100.00

[6] Renal and Urinary Affections.

Malenfant found that salivary calculi (located in Wharton's duct) was composed of:

Lime phosphate	27
Magnesium phosphate	1
Basic lime phosphate	60
Alcohol and muriatic acid	4
Ptyalin	2
Loss	6
	100

The following are results of analysis of salivary calculi by various observers:

Calcium carbonate	81.2	79.4	80.7	13.9	30	15	2
Calcium phosphate	4.1	5.0	4.2	38.2	75	55	75
Magnesium phosphate	.....	.....	.....	5.1	. .	1	. .
Soluble solids	6.2	4.8	5.1	38.1	5	25	23
Organic matter	7.1	8.5	8.3				
Water and loss	1.3	2.3	1.7	6.3	. .	. .	. .

Deposits in the tissues in gout are made up of soda and lime urates. In order to compare the calcic deposits in other parts of the body with the so-called serumal deposits upon the teeth affected with interstitial gingivitis, thousands of teeth were obtained from three dental offices which make a practice of extracting teeth. From these one thousand were selected at two different times, making two thousand teeth containing deposits direct from the tissues. These were submitted to a chemical analysis by J. H. Salisbury, at Rush Medical College, who reports as follows:

"The method which I employed in analysis of calcic deposits was as follows: The material was so selected as to be free as possible from salivary tartar and a weighed portion was dried at 100° C. This was then carefully incinerated and again weighed, and the difference calculated as organic matter. The residue after incineration was divided into two portions, A and B.

"A was used for the estimation of phosphates as follows: The ash was dissolved in nitric acid and the solution precipitated

with ammonium molybdate. The precipitate was washed, dissolved in ammonia precipitated by magnesia mixture and the precipitate of ammonia magnesium phosphate, washed, dried, ignited and weighed.

"In B, calcium and magnesium were estimated as follows: The ash was dissolved in hydrochloric acid and the acid just neutralized with ammonia water and sodium acetate added. It was then made slightly acid with a drop of hydrochloric acid and precipitated with ammonium oxalate. The precipitate of calcium oxalate was filtered off, washed, converted into calcium oxide and weighed. The filtrate was made alkaline, sodium phosphate added, and the precipitate of magnesium-ammonium phosphate collected, washed, dried, ignited and weighed. In case the phosphoric acid determined in A did not saturate the calcium and magnesium obtained in B, the excess of base was calculated as carbonate.

"The following is the composition of the calcic deposits on the roots of the teeth, according to analysis of April 18, 1898:

Water and organic matter	32.24
Magnesium phosphate	.98
Calcium phosphate	63.08
Calcium carbonate	3.70
	100.00

"Analysis of the calcic deposits on the roots of the teeth October 24, 1898, shows it to have the following composition:

Water	4.48
Organic matter	27.00
Calcium phosphate	72.73
Magnesium phosphate	4.91
	99.12

The composition of the alveolar process is as follows:

Organic matter: Gelatine and blood vessels	33.30
Inorganic matter:	
Calcium phosphate	51.04
Calcium carbonate	11.30
Calcium flouride	2.00
Magnesium phosphate	1.10
Sodium oxide and sodium chloride	1.26
	100.00

By comparing the tables of the composition of calcic deposits upon the roots of teeth with that of the alveolar process, it will be observed that there is very little difference. Tartar deposited from the salivary glands and calcic deposits upon the roots of the teeth must not be confounded since there is little in common between them. Tartar is the principal cause of local interstitial gingivitis commencing at the gum margin, while calcic deposits are the result of interstitial gingivitis and are always located upon the root of the tooth at the point of absorption of the alveolar process. The amount of calcic salts in the blood is very small as compared with the amount deposited upon the roots of the teeth and what is lost in the fluids around the teeth. The author [7] has stated that the calcic deposit upon the roots of the teeth was the absorbed alveolar process and not derived direct from the blood as has been suggested.

While nearly every kind of food taken into the stomach contains inorganic salts, every excretory organ of the body throws out a certain amount of these salts. Some of these organs excrete the salts in a pure state, while in others the salts are combined with acids or fluids peculiar to that organ. These salts differ in composition and quantity on different days, at different hours of the same day; differ at different ages of the same person and differ in persons of like age, on the same diet. No matter how careful the chemist may be in analysis, no two results will be exactly alike. For this reason, in tartar and calcic deposit upon the roots of teeth, two different analyses of the same deposits are cited. It is evident that while slight differences occur in the table, these are due chiefly to the character of the secretions. The kidneys and salivary glands clearly excrete most of the waste inorganic salts.

Since each excretory organ has its part in elimination of waste inorganic salts, it is clear that if one organ becomes tired or diseased, other organs have an extra amount of material to excrete. In any event, the blood becomes surcharged with waste inorganic salts. There is a class of patients with deformed jaws and irregular teeth, tonsil hypertrophy, mucous membrane, nasal bone and post-nasal space disorder, adenoids, arrest of the

[7] Endarteritis Obliterans. The Dental Digest, 1903.

facial bones. They are neurotics and possess degenerate structures. This class comprehends those whose nervous system is unstable and whose physical development is a departure from the race type. This unstable or tired condition may affect but one excretory organ. In most cases it affects all organs as well as the entire body. In these patients, especially in youth, does hypertrophy of the alveolar process take place and large deposits are observed upon the teeth. In this class may be placed rachitic children.

Inorganic salts taken in food are generally utilized until the osseous system has attained its growth. This usually occurs at about the twenty-sixth year, but full growth may not be attained until the thirty-sixth year. When this period has been reached, although the body still has the same supply of inorganic salts, the system can assimilate only what it needs. The remainder becomes waste. Under such conditions the blood is overcharged with these salts.

A condition of the system, which has received too little attention, occurs in a class of children ranging from six to eight years, who excrete larger quantities of inorganic salts through the kidneys and salivary glands. In such cases the teeth become coated with tartar. The gums become inflamed from irritation. Interstitial gingivitis is developed in youth. These children may be rachitic, or border upon the disease. They are neurotics, with degenerate structures, suffer from rachitis, rapid decay of the teeth and irregularities. They occur in American and European schools of idiocy and for dependent and defective children. From seventy-five to ninety per cent of these children have interstitial gingivitis, ranging from simple inflammation of the gums to absorption of the gums and alveolar process with pus exudate. Miller noticed in an examination of twenty-six cases of rachitic children under twelve years of age that seven manifested pronounced symptoms of interstitial gingivitis. This was no doubt due to accumulation of calcic salts upon the teeth, producing irritation and absorption of the alveolar process and contraction of the gums.

In cases where large collections of tartar are deposited upon the teeth of children there is also an excess of excreta through

the kidneys. Examination of urine in such cases will reveal always from four to eight times more deposit than the normal for the age of the patient. Defective nutrition is the result, the bones are small, and the jaws and teeth are irregular. The teeth decay early in life and it is with difficulty that the decay can be arrested. What is true of children is also true of people at advanced age.

After the skeleton had attained its growth (even in those cases where no deposits were before observed) the blood became overcharged with lime salts and the teeth became a nidus for the deposit from the salivary glands. It is, therefore, clear why deposits and inflammation of the gums are so common after the twenty-sixth year, and more common later in life. Defective children and people who have obtained their growth are more susceptible to trophic disorders of nutrition and the tissues take on disease more readily than healthy individuals earlier in life. When inflammation takes place in connective tissue in all parts of the body (especially if the blood be surcharged with inorganic salts) deposits take place in that tissue through the capillary system. On the other hand, when inflammation of the connective tissue takes place, if inorganic salts be scarce in the blood, deposits do not take place. As is elsewhere shown,[8] calcic deposits on the roots of teeth are a result of inflammation and pus infection and not the cause.

---

[8] International Dental Journal, April, 1896.

CHAPTER VIII.

THEORIES OF INTERSTITIAL GINGIVITIS.

The etiology of interstitial gingivitis, according to the views summarized previously, is divisible into local and constitutional. While one school leans largely to the local etiology, another advocates as strongly the constitutional theory, and a third believes in both the constitutional and local theories as causes. The author, from his elaborate researches which began in 1886, is an exponent of the latter class. In a general way, etiology may be divided into exciting and predisposing. Etiology may also depend upon an element dependent on the exciting cause, an element dependent on the constitution of the individual attacked, and finally an element dependent on his condition when attacked, both as regards his general system or any one of his organs. The chief constitutional causes to which the disease has been ascribed are general conditions of the health, heredity, constitutional disorders, excessive lime salt secretion, meat-eating, nervous exhaustion, scorbutus and uric acid states, as well as environment. To these may be added drug and metal poisoning such as mercury, lead, brass, arsenic, bromides, etc., as well as autointoxication. The local causes assigned are acute inflammation of the mucous membranes, catarrhal states, germs or fungi, irregular teeth, lactic acid, pocket disease, hemorrhagic deposits, serumal calculi and uncleanliness. That all these factors exercise an influence is undeniable, but the enormous etiologic role which has been assigned to some of them is the result of generalization from too few causes. Many of the assigned causes could be compressed into fewer etiologic influences. Thus meat-eating, the uric acid states, arthritis or gout are too intimately connected to be regarded as different causes, from a constitutional standpoint. As has been already pointed out, uric acid acts, when it acts at all, like lactic and other acids, as a local irritant rather

than as the constitutional condition (as many suppose) which underlies its production and of which it serves as an index.

Scorbutus is an expression of a nutritional disorder due very frequently in the adult to an excess of meat or a monotony of diet. It is a constitutional disorder, peculiarly apt to have its local expression in the gums long ere the general constitutional symptoms are manifest. The germs and fungi etiologists, on the other hand, tend to ignore the constitutional state behind the local culture medium, which must be furnished before growth of the germ or fungus can occur. In order, therefore, to determine whether an alleged cause be exciting or predisposing and what is the influence of the etiologic moment, as the union at one time of the two constitutional factors already cited is called, analysis is required of all the varied factors charged with producing the disease. The influence of heredity is generally left out of consideration unless it be direct, which it rarely is, since heredity, as has been well remarked, is usually a prophecy rather than a destiny. It hence constitutes, as a rule, a predisposition.

The chief tissues concerned in the elimination of waste products are the skin, the lungs and air passages, including the mouth and nose, the kidneys, liver and intestines. Interference with the eliminatory powers of the kidneys, liver and intestines causes autointoxication and is especially apt to throw extra work on the skin, lungs and air passages. Of this a sour-winey odor of the breath in diabetes is an excellent illustration. What is true of such a marked form of suboxidation, resulting in autointoxication, is true of less pronounced forms. The peculiarly foul odor of the breath and skin in fæcal intoxication indicates that the mucous membranes of the nose, throat, mouth and gums are doing the work of elimination which should have been done by the intestines. The failure of the kidney to perform its share of eliminatory work is most apt, however, to find expression in the skin, lungs, nose, mouth and gums.

The influence of the nervous system on the growth and repair of any tissue is admitted by every physiologist. This influence is entitled the trophic function of nerves. It is not, however, exactly settled whether it be exerted through the nerves themselves or secondarily through their control of the

vaso-motor (blood vessel) system. Many trophic disturbances, as J. Collins [1] remarks, are probably due to vaso-motor changes, and it is not possible to separate by any sharply defined line the vaso-motor from the tropho-neuroses. At the same time, it should be distinctly remembered that there exist tropho-neuroses in which there are no appreciable vaso-motor changes as in many cases of acromegaly and hypertrophies. On the other hand, there are any amount of vaso-motor disturbance which are by no means trophic in character. Trophic disturbance, which may play a very important part at the onset of interstitial gingivitis, is neurotic œdema due to nerve irritation. While this is most frequent on the face, lips, tongue, pharynx, forehead and genital organs, it also appears on the gums. The œdema reaches its full development in from one-half to two hours. There is a feeling of stiffness and unyieldingness, but no sensation of inflammatory swelling. This type of trophic disorder often initiates changes in the mucous membrane which may readily form the basis of interstitial gingivitis. This condition may not be only due to ordinary nervous causes, but may arise from constitutional conditions, gout, etc., and toxic influences.

---

[1] Nervous Diseases, by Dr. F. X. Dercum.

CHAPTER IX.

URIC ACID AND INTERSTITIAL GINGIVITIS.

Uric acid was first isolated by Scheele in 1776. It consists of a white spongy powder. It is devoid of taste and odor. Under the microscope it is seen as rhombic tables or as elongated plates resembling sheaves or rozettes. As deposited in the urine, it has a more or less reddish tinge due to the presence of urinary coloring matter.

The nitrogenous constituents of urinary excretion consist chiefly of urea or uric acid in certain animals and other nitrogenous urinary constituents.[1]

Uric acid is found abundantly in the urine of the lower vertebrates, such as reptiles, birds and mammals. It would be strange if it were not found in the fish tribe. It is more abundant in birds than in reptiles. It seems to be a normal constituent in both. Uric acid occurs more frequently in the urine of carniverous mammals, although frequently absent. While found in the urine of the herbivora, the quantity is often small and variable. Traces of uric acid are found in the organs of these animals such as the brain, heart, lungs, spleen, pancreas, while it is always found in the blood of birds. In birds, the uric acid is partly formed from the purin bases. It would be strange, therefore, if it did not develop in man since he has retained many of the phylogenetic peculiarities of his precursors. In human urine, uric acid is observed in variable amounts. It has been observed in healthy human blood. According to Hammarsten,[1] the amount of uric acid eliminated with human urine varies considerable but amounts on an average to 0.7 grams per day.

Hammarsten says, "We used to ascribe an increasing action upon the elimination of uric acid to proteid food, but the investigations of Hirschfeld, Rosenfeld and Orgler, Silven, Burian and Schur and others have positively proven that a diet rich in pro-

[1] Hammarsten, Physiological Chemistry, page 485.

teid does not itself increase the elimination of uric acid but only according to the amount of nucleins or purin bodies contained therein. The common statement that the elimination of uric acid is smaller with a vegetable diet than with an animal diet, when the quantity may be two grams or more per twenty-four hours, is explained by this." We would naturally expect to find uric acid in patients who live on a strictly vegetable diet as well as in those who live on a meat diet, since uric acid is found in herbivora as well as carnivora.

The uric acid, in so far as it is produced from nuclein bases, is, in part, derived from the nucleins of the destroyed cells of the body and in part from the nucleins of free purin bases introduced with the food.

Belonging to the same group as uric acid are hypoxanthin, xanthin, guanin and adenin. These are called purin bodies and are liberated during the digestion of nucleo-proteids contained in food. It has been found that a diet of meat, especially veal, liver, pancreas and sweetbreads containing a large amount of nucleo-proteid, leads to an increase in the excretion of purin bodies in the urine as compared with a diet of eggs, butter, milk, fruit, vegetables, cheese and bread. The amount of uric acid excreted in new born infants is in excess as compared with the adult.

The morbid conditions, in which the uric acid passed in the urine is increased, are leucocytosis and leukaemia. This increase may be attributed to the degeneration of the excess of leucocytes in the blood. Certain drugs increase the amount of uric acid, e. g., pilocarpin and salicylates.

Luxury and modern degeneracy are generally charged with the production of diseases which were later found to have attacked man in prehistoric periods. This has been the case with interstitial gingivitis.

R. R. Andrews expresses the following opinion as to modes of life: "I have been led to believe from my own experience that this trouble exists largely in the mouths of people accustomed to luxury—good livers, people about middle age who over-eat and under-work."

No method of living can be regarded as a cause of interstitial gingivitis except so far as it affects the general system, thus producing trophic changes. There is probably a slight difference in liability to interstitial gingivitis between people of sedentary habits and active outdoor workers, as well as between animals domesticated or in captivity and those which run at large.

It is, however, obvious from the data of the chapter upon "History" that all races and stations, regardless of time, climate, or mode of life, have suffered with the disease. Examinations of animals in the American and European zoological gardens show that it is not confined to any class of animals. Dogs, cats, horses, cows, whether housed or running at large, suffer with it as age advances.

Uric acid formation is not confined to large eaters. Spare eaters may have considerable quantities since they may be unable to take care of the uric acid derived from the moderate amount of uric acid forming substances (purin bodies) in a normal diet.

To summarize then, it will be seen that the age of the patient, the condition of his system, the character and quantity of food eaten and certain drugs must always be considered in relation to the quantity of uric acid excreted.

Since the discovery of uric acid in the urine by Scheele in 1776, the alleged influence of this factor was steadily advanced to the time of Haig, of London, the most prominent exponent of this theory. Since his time, a better knowledge of uric acid formation and its influence upon the system has revealed the usual exaggeration of the influence of this particular etiologic element. Researches have shown that uric acid poisoning, in a greater or lesser degree, is dependent upon the state of the system, the nature and strength of the exciting cause. All three play a part in the digestion, assimilation and elimination of the purin bodies which underlie the condition of health formerly attributed exclusively to uric acid.

The uric acid theory of disease having been so strongly advocated by certain physicians, a number of dentists have applied the same theory as a cause of interstitial gingivitis and pyorrhœa alveolaris.

What John Fitzgerald[2] calls the gingival organs, possess, as he remarks, in common with some other tissues of the body, the power of selecting and excreting poisonous substances from the blood. Some of these cause hyperæmia, or even inflammation, in their passage. Uric acid has been found to play a part in so many excretions that it has naturally attracted attention here. The trend of medical opinion has set strongly in this direction, but of late this trend is changing.

During the past two decades uric acid has assumed again the prominence in pathogeny which it once had when called suppressed gout. It is not surprising, therefore, to find that Reeves, Pierce, Rhein and others claim a uric acid etiology for interstitial gingivitis. In support of this claim are advanced the results of three experiments which Pierce has had made on tooth deposits. These deposits were examined chemically by Ernst Congdon, of the Drexel Institute.[3] The first specimen contained a number of needle crystals of calcium urate, a few crystals of free uric acid and crystals of calcium phosphate. Destructive distillation gave a strong ammonic reaction. The murexid test for uric acid and its compounds gave faint results, although its characteristic color was evident in several places. The second specimen presented the same crystals. The reaction to the murexid test was strong and resulted in a number of purplish-red spots. Similar results were obtained from the third specimen. A. B. Brubaker examined six or eight specimens in Pierce's presence, with like results to those obtained in the previous examination. In three an abundance of sodium urate crystals were present.

The great deficiency in the experiments thus described is the small number of cases examined and the lack of proper control experiments. These elements have so frequently led to errors in dental pathology that I determined upon a series of investigations in two different laboratories, whose results were reported some years ago.[4] The Columbus Medical Laboratory was selected for one series of experiments in special cases. The labora-

[2] The Clinical Journal, March 1, 1899.

[3] International Dental Journal, Vol. XV, pages 1, 217, 501.

[4] Dental Cosmos, April, 1896, page 310. Journal of the American Medical Association, January 16, 1897.

tory of the Northwestern University Woman's Medical School was selected for the other series of experiments, to which teeth were sent as soon as they were obtained. One hundred and fifteen teeth were sent to the laboratory last named from three institutions in Chicago which make a specialty of extraction. These teeth had no history other than the fact that the cases were well-marked instances of interstitial gingivitis with plenty of calcic deposits, and that the teeth were loose in the sockets when extracted. Of the one hundred examinations made in the Columbus Medical Laboratory, fifty were upon specimens of calcic deposits from my patients and fifty were upon specimens obtained from the institutions just mentioned, and were therefore without history. The tests employed were the hydrochloric acid, the dry distillation, and the murexid, these being the tests recommended by Pierce. The examinations in the Columbus Medical Laboratory were made by J. A. Wesener, and those in the laboratory of the Northwestern University Woman's Medical School by J. H. Salisbury.

Of the one hundred and fifteen examinations made at the Northwestern University Woman's Medical School by the first test, in only two cases were found the needle-shaped crystals, and one in which there was a slight resemblance of uric-acid crystals. By the dry distillation test, thirteen gave no reaction from ammonia, and in seven the reaction was slight. The remaining eighty gave a decided reaction. By the murexid test, four gave a slight murexid color, but remainder gave no reaction. Special examinations was made of twelve of these teeth by the addition of strong hydrochloric acid, warming, decanting the acid, and washing with water. These gave no reaction by the dry distillation for ammonia. Two gave a slight reaction by the murexid test. In examination of the teeth of three uric-acid diathetic women, over forty years of age, uric acid was not detectible either by the murexid test or microscopically. The examinations made in the Columbus Medical Laboratory were still more interesting, since among them were specimens from patients whose history could be obtained. Of the fifty obtained outside, eight gave positive results from all three tests. The other forty-two were positive by dry distillation, and negative by

the murexid and microscopical tests. Of the fifty patients, thirty-eight females and twelve males, thirty-two were over forty years of age, twelve over thirty years, and six over fifteen years.

Twenty-six have uric acid to a greater or less extent, nine suffer with indigestion, seven of which are subject to sick headache, thirty-four have rheumatism. Six are English, and four of these have the true gout; the other two have rheumatism.

All are positive with the dry distillation test. All are negative with the murexid test. Forty-nine are negative with the microscopical test. One shows needle-shaped crystals, but not uric acid. It is a singular fact that in both laboratories, the cases in which there were uric acid and gouty histories gave negative results. By the dry distillation test, out of two hundred and fifteen cases, all but twelve cases (which have been treated to remove nitrogenous material) responded. The twelve cases so treated did not respond, since nitrogenous compounds in and about teeth (even the saliva) burned to an ash will produce ammonia. By the murexid test only twelve out of the two hundred and fifteen gave a positive reaction. By the microscopic examination but ten showed crystals. One of the chemists who made the examination is positive that they were uric acid crystals. The other is not, since lime-phosphate crystals resemble uric acid crystals too minutely to be distinguished positively.

For three years Wesener made further examinations as to the relative value of the three tests employed. According to his experiments the murexid test is the most valuable, the crystal test second, and the dry distillation third. The murexid test is the most reliable in testing tartar for uric acid, since its red color is easily distinguished from other colors and the test is simple in application. The test for crystallized uric acid is very unsatisfactory, since here must be dealt with a complex mass which not only contains crystals of calcium phosphate (very similar to those of uric acid) but a great mass of detritus obscuring the crystals of uric acid. If crystals be present they by no means settle the existence of uric acid. When the faintest quantity possible of uric acid is mixed with tartar from teeth and subjected to crystallization, the results are always negative. If subjected

to the murexid test, the results are always positive. The dry distillation test is so inaccurate as to be unworthy consideration.

Since these results were published, seven hundred and thirty-five cases have been examined. These examinations were conducted by Jerome H. Salisbury, now of Rush Medical College. The teeth procured from institutions which make a specialty of extracting contained the dark calcic deposit above the pus line. By the murexid test, six out of the three hundred gave a distinct reaction; eighteen showed crystals under the microscope. The murexid test was performed as follows: The deposit was selected as carefully as possible, removed from the tooth, and placed in a small porcelain crucible. A drop of pure nitric acid was added and the mixture evaporated on the water bath. When dry, the evaporation was repeated with another drop of nitric acid, and the crucible allowed to cool. When cool, the color produced by the nitric acid was observed, and then a glass rod, wet with ammonia water, was brought near the deposit, and any color produced was noted. If no color was observed, the ammonia was allowed to flow over the residue. A yellow color was produced in many cases by the nitric acid, which was deepened by the addition of ammonia. The microscopic examination was made by scraping off the deposit and evaporating it with a drop of hydrochloric acid. The residue was moistened with water, and the insoluble material placed on a slide and covered with a cover-glass. It was examined with a No. 7 objective. Uric acid, therefore, occurred in a certain very small proportion of cases of calcic deposit on the teeth.

Four hundred and thirty-five cases were later examined, making in all nine hundred and fifty. Out of these four hundred and thirty-five cases only four per cent showed uric acid by the murexid test and eight per cent by the crystal test. Since the crystal test is not so accurate as the murexid test, it is safe to say that six per cent was the actual per cent of uric acid. As a result of the different experiments, in the first two hundred and fifteen cases five per cent uric acid was found. In the second three hundred cases, four per cent, and in the third four hundred and thirty-five cases, six per cent was found. In an examination of nine hundred and fifty cases by different chemists at different

periods, five to six per cent give positive results as to uric acid by the chemic and microscopic examination. These results demonstrate conclusively that interstitial gingivitis is not due solely to uric acid; that uric acid when found is merely an expression of the uric acid diathesis and a coincidence, since it is not always present in the gums and tartar of patients attacked either by gout or the uric acid diathesis. In the six per cent of cases there was nothing to show that uric acid was the cause of interstitial gingivitis, since the deposits were examined after the teeth had been removed. Any other irritation may have been the exciting cause. Uric acid acts, when at all, solely as a local irritant, like other acids and poisons. A microscopic examination of the tissues involved occasionally reveals uric acid crystals. The fact, however, that they are found in a small number of patients suffering with interstitial gingivitis shows that they cannot be depended upon as a general cause of the disease.

CHAPTER X.

HEREDITY AND ENVIRONMENT IN INTERSTITIAL GINGIVITIS.

The relations of heredity are far more intricate than is usually assumed to be the case in the average discussion of the subject. The problem consequent on impregnation is not that involved in the mere carrying of the mixture of parents in a fully developed form through intra-uterine life. As all vertebrate organs pass through the same stages before definitely differentiating, the later types have to gain at the expense of the earlier and hence must receive greater energy from the direct ancestors. The want of this energy is shown in the various defects and departures from types which occur in the different degeneracies and congenital defects. The types of heredity ordinarily considered are direct heredity where the individual takes after immediate ancestry, and type heredity where he takes after the type to which he belongs.

The influence of heredity in interstitial gingivitis, as in other morbid conditions, is still a mooted question. Morbid heredity, as I have elsewhere shown,[1] is practically divisible into direct and indirect. The first is the direct inheritance of the weakened organism of the mother; the second is a condition of intra-uterine infection. Heredity further should be distinguished from congenital states which result from the operation of germs or toxins during a particular pregnancy wherein these pass through the placenta to the fœtus. A child may be born of a tuberculous mother with a tendency to tuberculosis but may not develop it; on the other hand the tubercle bacilli may infect it through the placenta so that it is born with tuberculosis.

The weakened organs of the mother (due to an unstable nervous system) may cause the child to inherit an unstable nervous system. This, in turn, may cause an arrested or excessive development of the jaw and alveolar process in the child at the periods

---

[1] Talbot. Degeneracy: Its Signs, Causes and Effects.

of stress. Under such conditions the jaw is most frequently arrested. An arrested jaw and alveolar process usually mean an irregular dental arch in which the teeth are so closely packed (owing to the crowns being straight, not bell-shaped) that the alveolar process is almost entirely destroyed between the teeth. To keep the gum margins clean and in a healthy condition in such a mouth is almost impossible. The slightest inflammation, due to irritation, of the gums, peridental membrane or alveolar process, whether local or constitutional, will cause its destruction sooner or later in the life of the individual.

Again, because the alveolar process is a transitory structure as well as an end organ, and because of its thinness around the teeth, the inheritance of a lowered vitality, both in the individual or in the immediate structure under discussion, or both, will furnish a predisposing cause for interstitial gingivitis. This is why so many neurotic and degenerate children possess irregular jaws and teeth, and why these children early develop interstitial gingivitis.[2] Hence heredity may well be the indirect cause of interstitial gingivitis.

The reported cases of direct heredity in the pyorrhœic stage of interstitial gingivitis may belong in one or the other of these categories, but such a theory can hardly be considered tenable. Researches of many able investigators have failed to demonstrate the germ theory, local or inherited, in causal relation to interstitial gingivitis, as practiced by specialists, as we shall see in Chapter XII. It is known, however, to every specialist that the *secondary condition* of this disease (pyorrhœa alveolaris) is due to germ infection. Pus germs producing the secondary stage of the disease, are local in nearly every mouth and are not supposed to be inherited. Pus germs in the mouth do not, apparently, produce the *first stage* of the disease (interstitial gingivitis), which is simple inflammation. We could hardly expect to find directly inherited germs from the mother to the child causing infection of the gums, peridental membrane or alveolar process later in the child's life, since there is a period between birth and the eruption of the temporary teeth where no alveolar process is present and no infection takes place.

[2] Talbot. Developmental Pathology: A Study in Degenerative Evolution.

Transitory organs are bound to be weakened by heredity, both in their structure and in their resistance to morbific germs and agencies. These weaknesses are especially apt to be emphasized during the second and third periods of stress[3], when the temporary and permanent teeth are erupting. Such weaknesses may be the outcome of general nerve exhaustion on the part of the parents (the mother especially) or of the child itself, and they represent a changed (transformed) heredity far more commonly than a direct. This heredity may be more intense than the constitutional lack of health in the parents. On the other hand, the influence of intermarriage of several healthy generations may so offset the evil results of the defects in the parents that the inheritance of disease or the tendency to disease is slight, if at all existing in the child. The last type of heredity, called atavism (or "throw-back" by breeders), is more likely to work for good than for evil, although disease effects are more generally looked for. Concerned in this latter, where the individual throws back to immediate remote ancestors, this element of atavism tends, through preserving the type, to offset the defects of immediate heredity and, indeed, often underlies the apparent difference between children of the same parents. It likewise prevents equal inheritance from both parents, and sometimes favors inheritance of strength or defect from either. It underlies also so-called collateral or indirect heredity and the transmutation of heredity. By virtue of this atavism, a serious nervous defect in a parent or parents might express itself only in an increased tendency to disease on the part of the child's transitory structures and end organs.

The periods of stress are times in the life of man when certain great life functions are developing or undergoing retrogression. These periods of stress are, during development in utero, during the first dentition, during the second dentition (often as late as the thirteenth year), during puberty and adolescence (fourteen to twenty-five), during the climacteric (forty to sixty), when uterine involution occurs in woman and prostatic involution in man, and finally, during senility (about sixty and upwards). These periods often constitute a cause for the produc-

[3] Talbot. Developmental Pathology: A Study in Degenerative Evolution.

tion of disease even though hereditary defect itself be absent until this time when it first makes it appearance.

Another factor to be considered in this connection as complicating the diagnosis of heredity in interstitial gingivitis is environment, understanding by this term all the external conditions that can favor the development of the disorder. Family habits and surroundings are apt to be alike for every member, so that if anything in the environment especially favors the breaking out of a disease in one member, the same cause or causes are equally likely to favor the occurrence of the disorder in several members or even generations of the family, and this may give rise to a suspicion of heredity. This consideration applies to interstitial gingivitis, since the disease has been known to develop in different members of the same family at similar periods of life and under the same conditions.

That constitutional conditions of hereditary origin favor the occurrence of interstitial gingivitis is undeniable, but this does not mean that interstitial gingivitis itself is hereditary. They favor its occurrence just as they favor any other morbid condition, by lessening resistance or by preparing the way. The interstitial gingivitis is only one of the many accidents that are thus facilitated.

So far as salivary concretions are to be regarded as an exciting cause, heredity may be put out of court, since these (though varying widely in different individuals in the amount of the deposits, and consequently in the irritation produced) are dependent upon more remote constitutional or local conditions and have no direct connection with the heredity. Thus the various deposits attributed to lithæmia or arthritic conditions (notoriously hereditary), are merely incidental to those conditions and not essentially connected with their constitutional origin. The constitutional conditions merely happen to furnish the irritant.

Local uric acid poisoning[4] is, as I have elsewhere shown, occasionally associated with interstitial gingivitis. The coexistence signifies the lowered vitality of the system and autointoxication, rather than the etiolgy.

---

[4] The Dental Cosmos, 1896, page 312.

The same is true of all the other neurotic, rachitic and degenerative conditions, hereditary or otherwise, that are met with, associated with gingival inflammation. They favor the occurrence of the disease by causing a weakened capacity of resistance, thus predisposing to the attack of any irritation. The mouth, resistant as it ordinarily is, is at all times open to irritation and infection. When resistance is impaired it gives way at its most vulnerable points, and the gingival margin because of its transitory and end organ nature is one of these points. Interstitial gingivitis is favored or hindered, like other disorders, by constitutional conditions which may or may not be inherited, and which bear toward it the relations only of predisposing and accessory causes.

To summarize, therefore, it is reasonable to suggest that interstitial gingivitis and pyorrhœa alveolaris are not inherited.

Interstitial gingivitis is the primary condition and represents the reaction of a weakened transitory end organ to constitutional or local irritation.

Heredity, direct and indirect, may of course weaken resistance and predispose to the disease, particularly in a structure so transitory as the alveolar process, but no direct transmission of infection is either demonstrable or even tenable.

Pyorrhœa alveolaris is a secondary infection grafted on the original inflammation by the agency of local pyorrhœic germs such as are prone to invade any exposed membrane, and have no relation whatever to heredity.

CHAPTER XI.

DEGENERATE TISSUES IN INTERSTITIAL GINGIVITIS.

One important factor of predisposition to interstitial gingivitis is degeneracy, either local or general. Three possibilities of life await each living being. The individual may remain primitive and unchanged, progress toward a higher type or retrogress to a lower type. In these three conditions, the factors underlying the stable state force the structures to remain as they are; those underlying the progressive tendency make them more elaborate, while the third tends to simplify structure. Degeneracy is a gradual change of structure by which the organism becomes adapted to less varied and less complex conditions of life. It is a reverse of development which proceeds from the indefinite and homogeneous to the definite and heterogeneous with a loss of explosive force due to the acquirement of inhibitions or checks. In proportion to the depth of degeneracy does it affect the early simpler or late complicated acquisitions. The opposite process of progression is a gradual change of structures by which the organism becomes adapted to more varied and more complex conditions of life. In progression there is a new expression of form corresponding to new perfection of work in the animal machine. In degeneracy, there is suppression of form corresponding to cessation of work. Elaboration of some one organ may be the necessary accompaniment of degeneracy in all the others. On the other hand, degeneracy in one organ may be the necessary accompaniment to elaboration in all the other organs. During any of the periods of stress defects due to degeneracy are apt to appear and affect the line of least resistance, determined by the depth of degeneracy, as well as the variability of the structures concerned. This is the reason certain individuals develop disease or become susceptible to disease since at these periods the entire organism undergoes change, and the organs most affected by degeneracy are the

first involved. This is particularly true of transitory structures like the alveolar proccess and it, in addition to being doubly transitory and the most sensitive structure in the body is also an end organ like the brain, eye and kidney. The teeth and jaws are among the most variable structures in the body, and they are peculiarly apt to be affected by either general degeneracy, which affects the body as a whole, of local degeneracy, which may affect one organ or structure or part of them. Degeneracy factors causing nervous exhaustion in the parents leave their stamp on the tonicity of the child's organs to combat disease.

Every nerve cell has two functions, namely: sensation or motion and growth which are dependent upon each other, that is, if the cell be tired by excessive work along the line of sensation or motion, growth later becomes impaired. The cell then not only ceases to continue in strength, but becomes self-poisoned. Each of the organs (heart, liver, kidneys, etc.) has its own system of nerves (the sympathetic ganglia) which while under the control of the spinal cord and brain, act independently. If these nerve centers become tired, the organ fails to perform its functions, the general system becomes both poisoned and ill-fed, and nervous exhaustion results. In most cases, however, the brain and spinal cord are first exhausted. The nerves of the other organs are thus allowed too free play, and exhaust themselves later. This systemic nerve exhaustion particularly affects the testicle in the male and the uterus and ovaries in the female, hence an unstable nervous system in the offspring results. Through this, the body is imperfectly supplied with natural tonics (antitoxins) formed by these structures, and the general nervous exhaustion becomes still more complete. All the organs of the body are thus weakened in their function. Practically the neurasthenic's organs have taken on degenerative functions though not degenerate in structure. Through the influence of these various nerve exhaustion agencies, the spinal cord and brain lose their phylogenetic gains and the neurasthenic is no longer adjusted to environment. Since the reproductive organs suffer particularly, children, born after the nervous exhaustion in the parents, are more or less checked in development owing to the depth of degeneracy and the influence of healthy atavism.

They have degenerations in the structure of their organs which, in the parents were represented by neurasthenic disorder in function. As the ovaries of the neurasthenic female generally exhibit prominently the effects of nervous exhaustion, the offspring does not retain enough vigor to pass through the normal process of growth or should it survive, it is usually affected by the profound neuroses. In these instances in connection with irregular dental arches, there is always interstitial gingivitis and pyorrhœa alveolaris.

The action of degeneracy, considered as a local factor of constitutional origin, may be exerted to preserve embryonic conditions in adult life. Such preservation may result in the breakdown of tissues which would otherwise withstand germs, toxins and poisons, or other causes of disease external to the tissues. Given this condition of local degeneracy, a local predisposing factor is added to both the exciting causes and the constitutional predisposing factors. So long as the teeth and transitory structures remain in the comparatively stable condition of primitive races, this factor is, to a great extent, in abeyance. When, however, the jaw begins to evolve (grow smaller), the degenerate types find this factor adding dangers in their phylogeny. In the degenerate, the struggle for existence between organs (the brain and skull on the one hand, and the face, jaws and teeth on the other) is not properly balanced, whence the dangers from these local states of degeneracy that in the higher types are expressions of advance undergone without danger. This is excellently illustrated in the embryology of the mucous membrane. This, in degenerate children, often fails so to develop that the bactericidal function of mucus does not appear. This hereditary feebleness of the mucous membrane is peculiarly apt to occur in the nose, throat and gums, but other mucous membranes are not exempt.

Miller, as elsewhere stated, found a little over thirty-three and one-third per cent on examination of twenty-six rachitic children under twelve years who manifested interstitial gingivitis. Considering that most of these manifested symptoms of inherited congenital or acquired constitutional defect, such a small proportion is rather remarkable. The fact suggests one

of two explanations—either the children in the institution visited by Doctor Miller took better care of their teeth and gums than is usual with this class, or the cases in which pus existed only were classed as pyorrhœa. I have examined the mouths of deaf mutes, blind, idiotic, feeble-minded and rachitic children in the institutions in America and Europe. Interstitial gingivitis was found in all its stages, from simple inflammation of the gums to loosening of the teeth, in from twenty-five to seventy-five per cent. In these cases not only are there constitutional factors, but also uncleanliness of the mouth and gum tissues. The degenerate children of even the best families encountered in office practice usually have jaw deformities and teeth irregularities as well as interstitial gingivitis. Patterson has had under observation thirty-eight cases of well-marked pyorrhœa, thirty-three of which co-existed with nasal catarrh. These cases were, no doubt, those of degenerate patients. The nasal catarrh was a coincidence dependent on the general deficiency of the mucous membrane.

CHAPTER XII.

BACTERIOLOGIC RESEARCHES IN INTERSTITIAL GINGIVITIS.

The causes which produce interstitial gingivitis may be divided into those producing infections and those producing irritations from intoxication.

The infections may be divided into local and constitutional. The constitutional infections are those which infect the gums, alveolar process and peridental membrane, through the blood, such as tuberculosis, syphilis, scurvy and similar diseases.

The local infections are those in which the germs infect the tissues directly producing anthrax, actinomycosis, gonorrhœa, syphilis, apthae,[1] dead pulps and many other diseases for which no name has yet been applied.

All these diseases have constitutional symptoms which are associated and which must be understood in making a diagnosis. Since most of these infections, whether constitutional or local, are special diseases of the gums and alveolar process, they require special constitutional treatment according to the general symptoms as they arise and are therefore to be placed in a special class by themselves. They are not considered again in this work.

Experiments with bacteric infection upon animals and human to produce interstitial gingivitis have been made by Galippe, Miller, Rhein, Carpenter and Talbot.

Galippe[2] was probably among the first to make analytic experimentation in the bacteriology of this disease. He claims that there is found in the pus of pyorrhœa a parasite, resembling in shape the Greek letter N. Injecting this into the belly of a guinea pig, abscesses resulted, which had a special tendency to affect bone tissue. Injections into the space between the teeth and gums were negative in result. Galippe regards his experi-

[1] Talbot. Some Bacterial and Non-bacterial Diseases. Journal of the American Medical Association, February 10, 1912.

[2] Die Infectiöse Arthro-Dentäire Gingivitis, 1888.

ments as suggestions for further research, but not demonstrative.

Miller,[3] after explaining his own methods, made a series of culture experiments on agar-agar at blood temperature. Twelve cases of pyorrhœa in human beings, and six in dogs, were examined. He isolated twenty different bacteria from human beings, and nine from dogs. Among the twenty kinds, staphylococcus pyogenes aureus was found twice, staphylococcus pyogenes albus once, streptococcus pyogenes once. Of the other sixteen, nine subcutaneously injected produced no particular reaction, four a slight, three a severe suppuration in the subcutaneous connective tissue. . . . Among the nine species found in dogs, staphylococcus pyogenes albus occurred once. Of the other eight, two subcutaneously injected caused no reaction, and five but slight. One caused very profuse suppuration, by which large portions of skin exfoliated. . . . Microscopic examination of stained sections revealed masses of different bacteria, cocci and bacilli. Leptothrix occurred infrequently, and then only on the surface of the cement, and where there were microscopical cavities in it. . . . Miller succeeded consequently in cultivating a large number of bacteria from pyorrhœa alveolaris which possessed pyogenic properties, but was not able to determine the constant occurrence of any one which might be regarded as the specific micro-organism of pyorrhœa alveolaris. Miller remarks that it is not evident from Galippe's communication whether he found the N or B bacterium in all cases examined, or but once.

Sudduth, after repeated examinations, arrived at the same conclusion as Miller.

Dr. M. L. Rhein's[4] investigations are here given. He says, "The results of my investigations made at the Pediatric Laboratory in New York some eleven years ago are given here for the first time as corroborative evidence that in healthy bodies it is impossible to produce this disease. Four guinea pigs, proven later at the autopsy to be absolutely free from any taint of disease or abnormality, were chosen. Their food was carefully re-

[3] Micro-Organisms of the Human Mouth.

[4] Pyorrhœa Alveolaris. The Items of Interest, June, 1910.

duced day by day until they died at the end of ten weeks. Control pigs were kept in a cage alongside of these and fed with the usual quantities of food. About the beginning of the tenth week when the pigs were weakened from lack of food, all eight of them were inoculated in the pericemental regions with injections of liquid cultures developed in bouillon, from pus taken from the pockets of pyorrhœal patients. On the third day all evidence of the trauma produced by the injections had disappeared. At no time had there been the slightest evidence of even a resulting gingivitis. As stated before, the autopsies showed all four pigs to have absolutely normal organs, although they were practically skeletons. Of the control pigs, while three of them showed the same immunity to infection, the fourth one showed evidence of inflammation, and at the end of the fifth day there was a distinctive serus exudate coming from the neck of the front tooth around which the injections had been made. I have always regretted the fact that this pig was not killed and an autopsy held.

"Before this time I had examined the mouths of many hundreds of guinea pigs inoculated by the Board of Health with tuberculosis. Every one of these showed the most marked evidence of pyorrhœal conditions. In the same manner a visit to any sanatorium for tuberculosis cases will show on examination of the mouths, pyorrhœa alveolaris in a degree of severity exactly conforming to the inroads which the disease has made. A like examination of the medical wards of any hospital containing cases of diseases of the heart, kidneys, liver, lungs, etc., will, if no attention has been paid to prophylaxis of the mouth, show conditions of pyorrhœa. Even when the most strenuous efforts in this direction of mouth care are taken, if the form of the malnutrition has passed to a certain stage, no care of the mouth is sufficient to prevent the marked development of pyorrhœa alveolaris."

The results obtained in the Columbus Memorial Laboratory of Chicago, by W. A. Evans for the author, were as follows:

In order to determine whether a specific bacterium existed in the pyorrhœic stage of interstitial gingivitis in man (necessary to constitute this stage a special disease), pus from more than

fifty cases was examined. In all, the pus was obtained from the gums by a platinum needle under proper methods of sterilization. The pus from some cases was smeared on a slide. This was stained and such determination made as was possible with this procedure. With the pus from fifteen cases, agar was inoculated and placed in Petrie's dishes. The individual colonies were grown on gelatin, agar, bouillon, potato and blood serum. The results were as follows: In fifteen cases in which the organisms were plated out, fifty-five organisms were found. In two there was no growth. Two had but one species of germs, two had six, one had seven, and one had ten. The germs found are divisible into three classes: Those usually pathogenic to man, those exceptionally pathogenic to man, and those never pathogenic to man. The first class was found thirty times, the second twelve, and the third thirteen. Class third is, no doubt, seemingly smaller than it should be, since many members of it probably do not grow on ordinary culture media. Of the germs most frequent and important, staphylococcus pyogenes aureus occurred nine times, staphylococcus pyogenes albus six times, and staphylococcus pyogenes citreus once. A lanceolate diplococcus, growing like pneumonococcus, was found six times. Streptococcus pyogenes was found twice. Bacillus coli commune was found twice. A bacillus growing like the diphtheria bacillus occurred twice. This last bacillus had the appearance of the Klebs-Loeffler bacillus. It lay on the slide like it and it stained irregularly. Of the less important organisms, bacillus pyocyaneus was found three times, micrococcus tetragenus seven times, leptothrix seven times, bacillus mesentericus twice, bacillus subtilis three times. There was also present a peculiar large club-shaped fungus somewhat resembling the degenerative forms of actinomycosis.

Did these examinations stand alone, definite conclusions could not be drawn from them. These, however, are admissible since all observations on this subject tend in the same direction. While, as already stated, Galippe believed that he had isolated two bacteria capable of causing pyorrhœa alveolaris, still he failed with both to produce the disease. This failure, according to the laws of Koch, is fatal to the position taken.

M. Herzog, of the Chicago Polyclinic, on examination of cases of interstitial gingivitis, which had not reached the pyorrhœic stage, had the following results: Pieces from the gum margin which had been fixed and hardened in a formalin solution, were partly imbedded in celloidin, partly in paraffin. The sections were stained according to various methods, including Gramm's, eosin (Unna's) and alkaline methylblue stain. The examination of the tissue shows an unchanged lining of stratified squamous epithelium, and, in the connective tissue below the former, well-marked evidences of an inflammatory process. The round-cell infiltration is best marked in the deeper layers toward the periosteum, while the layers of connective tissue fibers nearer to the lining epithelium show less evidences of inflammation and are partly entirely free from any round-cell infiltration. The infiltrating round cells are of the type of lymphocytes, plasma cells and plasma mast cells. Very large and typical mast cells are frequently found in the neighborhood of small vessels. Many of the vessels seen are quite tortuous, and the vascular supply of the connective tissue appears to be considerably increased beyond the normal. Bacteria could not be demonstrated in the inflamed areas.

M. Herzog's examination of the interstitial gingivitis, produced by mercury in dogs, failed to reveal any bacteria. He was of opinion that the histologic changes of inflammatory type found, were due to the chemotactic influence of mercury and not to microbic action.

In a paper [5] read before the Section on Stomatology of the American Medical Association, at Columbus, Ohio, George T. Carpenter mentioned some very interesting experiments in this connection. By infecting a fresh wound in the gums of rabbits with pyorrhœa and other pus he found the parts will remain infected only from two to five days. In other rabbits a rubber band was placed around teeth and pressed under the gums until inflammation resulted, when the parts were infected with pyorrhœa and pus from a chronic ulcer; pus infection resulted.

Like experiments were made in the human mouth on gums

[5] Some Points on the Etiology, Pathology and Treatment of Persistent Pyorrhœa Alveolaris.

which had been neglected as well as on healthy gums, and with similar results. His experiments tend to show that, when animals and man are healthy, the tissues resist infection; but when diseased, infection results. All yield to treatment.

On examination of pus taken from pyorrhœa pockets proceeding from acute infection, two competent bacteriologists were unable to find a micro-organism not found in pus from other infected tissues.

These results, in Carpenter's opinion, tend to show that a specific germ, to which pyorrhœa alveolaris is attributable, has not yet been found.

The disease being so prevalent among dogs, it occurred to me that they would be of great value for experimental inoculation. The prevalence of the disease in dogs suggests that if it were a specific infection, these must be inoculable. Miller [6] had made a few inoculations of pus as well as of the deposits around the teeth. Slight inflammation, and, in one case, a little suppuration alone resulted. He afterward isolated twenty different bacteria from the human mouth and nine from dogs. Some of the uncommon varieties were infective, but without marked results. Isolated varieties would probably not produce results that could be attained by inoculating animals with the fresh secretion (pus and other deposits) from dogs already affected with the disease. A dog was procured from the Veterinary Hospital whose gums and outer alveolar process were almost entirely absorbed with pus exudate. Street dogs selected for inoculation were forty-six in number, ranging in age from one year to seven. They were of all breeds and conditions. Some were well fed, others very thin. Many had sound, healthy gums; others had slight inflammation at different localities. No dog was used whose gums and alveolar process had become infected or whose tissues were absorbed. Two dogs were operated upon at a time. The gum was separated from the necks of the teeth down to the alveolar process and peridental membrane—one half at the canine, the other at the second pre-molar, since in a majority of cases the disease began at the canine tooth, probably on account of its prominence and the thinness of the alveolar process. The sec-

[6] Micro-Organisms of the Human Mouth, page 329.

ond pre-molar was selected because it is the least prominent. The secretions about the teeth and gums of the diseased dog were collected upon a platinum wire (previously sterilized) and conveyed to the injured parts. Thirty-nine healed in eight days. In these the gum tissues were healthy. The pus had no effect. The wounds healed as rapidly as any wounds possibly could. In seven the gums were inflamed and infection occurred. Suppuration was slight in four and considerable in three. The pathologic findings in these cases were not unlike inflammation and infection in other tissues. Similar results would, no doubt, have taken place if inoculation had been performed with pus from an abscess. The last three dogs were allowed to depart at the end of four weeks with slight pus infection.

Since these researches were concluded, another series of experiments was undertaken upon the lines conducted by Dr. Carpenter, extending over a period of twelve years. These experiments were performed upon dogs, guinea pigs, rabbits, white mice, and humans. One hundred and seventy-six experiments were made. They consisted of the application of pus from pyorrhœa patients direct to healthy and diseased gums with a view of producing interstitial gingivitis or pyorrhœa alveolaris or both. Applications were made to fifty-six humans, twenty-six had slightly inflamed gums and thirty comparatively healthy gums. Forty-eight dogs were treated in like manner; twenty-nine young dogs had healthy gums and nineteen older dogs slightly diseased gums; thirty-two guinea pigs; eighteen rabbits and twenty-two white mice. While a slight inflammation was produced in a part of those cases where inflammation already existed, no extended chronic inflammation resulted. We would naturally expect to obtain some results in the white mice since they are of the degenerate type and very susceptible to disease but the results were similar to those of other animals. These experiments may later seem crude and with improved methods better results may be obtained.

Outside of a few specific diseases of the gums and alveolar process some of which have already been enumerated, no one has demonstrated that specific pathogenic bacterial infection is a cause of interstitial gingivitis although the mouth is known

to be the breeding ground of an extensive variety of germs. Teeth are extracted, irregular teeth are corrected, healthy gums are injured in filling teeth and finishing fillings many times each day but we seldom see local infection of the gums and alveolar process, producing interstitial gingivitis which comes to us every day for treatment.

In these diseased conditions, mechanical specialists spend hours on the surgical treatment of the gums, peridental membrane and alveolar process surrounding the tooth without first destroying the pus germs about the teeth, injuring the parts and carrying the pathogenic bacteria into the wounds. If infection were a cause, such rash treatment surely would intensify the disease. Some of the lower vertebrates, such as the carnivora live upon putrid food containing all forms of pathogenic bacteria but gum infection rarely, if ever, takes place.

Within the past year a machine has been placed upon the market for the supposed purpose of forcing oxygen through the tissues in the treatment of this disease. I have watched this process of treatment "with fear and trembling" since the method of application forces the pus germs through the inflamed alveolar process. Why infection does not occur is a mystery. This method of applying drugs and forcing pus germs into the tissues without infection is a strong point in favor of the non-infectious theory of interstitial gingivitis.

From our knowledge based upon original researches at the present time, interstitial gingivitis, from the viewpoint of the specialist, cannot be classed as an infectious disease. It may be possible in the future with more improved methods of research to throw a clearer light on the nature of the process.

CHAPTER XIII.

INTERSTITIAL GINGIVITIS.

Interstitial gingivitis is an inflammation which may take place in the gums, peridental membrane and alveolar process at any point from the gingival border to the apex of the root or roots of the tooth. This inflammation may be confined to a very small area at any one of these localities and progress to abscess and be restored to health without other parts becoming involved or the entire structure may become diseased resulting in the exfoliation of the tooth.

To illustrate, among the local causes tartar or other irritants at the gum margin will set up a gingivitis. Remove the irritant and by local treatment the gums are restored to health. The interstitial structures have not been involved. In autointoxication, poisons circulating in the blood may and do collect in the arteries midway between the gum margin and the apical end of the root, set up inflammation and a peridental abscess forms discharging directly upon the gum. The parts heal with very little or no pain to the patient. The gum margin or the apical end of the root or even the opposite side of the alveolar process is not involved. Here we have an interstitial inflammation. Again the pulp in a tooth dies, inflammation takes place at the apical end of the root and proceeds to abscess. Here again we have an interstitial inflammation without gingivitis. It is impossible, in many patients, to state just when the inflammation begins and in most patients it is both gingival as well as deep-seated. The term "interstitial gingivitis" is used to cover all the inflammation of the gums, peridental membrane and alveolar process.

Before discussing interstitial gingivitis we must first familiarize ourselves with the nature of the structures involved since there are no other structures in the human body like them by which the pathology can be compared. The structures and their functions are unique in themselves. We must, therefore, re-

capitulate briefly the main points already brought out in previous chapters in regard to the structures involved.

In the evolution of the face, jaws and teeth the tendency is for these structures to grow smaller, hence the jaws and alveolar process are transitory organs. In this natural evolution the influences of the parents, owing to excesses tend to produce tired out reproductive organs; these fagged out reproductive organs in turn tend to produce an unstable nervous system in both mother and child. The most intense effect is on structures which are transitory and always tend toward the least resistance.

After birth, the child is subjected to one or all the children's diseases which also tend to produce an unstable nervous system in the child. The effect of an unstable nervous system upon the child is to produce an arrest of development of transitory structures and acts also towards the line of least resistance. In both conditions, that of the parents and that of the child, the tendency is to produce a still smaller jaw and alveolar process.

Again, in some of the lower vertebrates, there is a succession of teeth throughout life. In the higher vertebrates, including man, there are only two sets; one is shed as soon as it has served its usefulness by absorption of the alveolar process and a second set takes its place. As soon as the alveolar process has built itself about the teeth, the phylogenetic influence is ready to remove the second teeth. In other words, the alveolar process is only waiting for some irritant to set up a low form of inflammation to produce absorption of the bone. We, therefore, have in the alveolar process a doubly transitory structure. We have also in the alveolar process an exceedingly sensitive end organ. The tooth to all intents and purposes, so far as this disease is concerned, is a foreign body. The arteries and nerves pass through the bone in a tortuous manner as far as the root of the tooth. Poisons circulating in the blood pass to the end of the arteries and stop setting up irritation and inflammation.

There are other end organs in the body but they are all composed of soft tissues. These tissues can and do expand when inflammation is set up and in many instances recover health. Not so with the alveolar process. When inflammation takes place in

this tissue, expansion of the arteries cannot take place and destruction occurs. The process rarely if ever recovers its lost tissue. Because the alveolar process is a transitory structure and end organ, it is the first structure in the body to respond to poisons and toxins. Two illustrations are only necessary to prove this statement. When a patient who has been working in metals or drugs visits his physician for any ailment, the first thing the physician does is to examine the gums to ascertain if the patient is poisoned by the drugs or metals, this structure being the first to become diseased and register the poisons.

Again, when the physician administers mercury or potassium iodid in the treatment of a specific disease, he continues the treatment until the gums are ''touched'' (inflamed), this symptom being the first and only sign that the system is under the influence of the drug. In lead poisoning we have the blue gum; in mercury, red; brass, green; scurvy, red, etc. The colors are due to the kinds of poisons collected in the ends of the capillaries next to the root of the tooth. These indications are sufficient proof of the statement that the alveolar process is the first structure involved.

INFLAMMATION.

It is not my intention to enter into a minute description of the phenomenon of inflammation but to simply state as briefly as possible how it is produced and the changes which take place in the tissues involved in the disease under discussion.

The most simple illustration of active inflammation in a highly vascular tissue is that of an injury to the fairly transparent web of the hind foot, tongue or mesentery of the frog. The web of the foot of a small frog is so thin that the changes occurring in and around the vessels of the part injured can be readily found with the microscope.

With slight modifications due to local conditions in the tissues under examination, the process of inflammation is the same throughout the entire vertebrate series from the reptilia upwards.

The frog is prepared by destroying the central nervous system or spinal cord by passing a wire through the vertebral col-

umn after first being curarised. The web of the foot is then placed under the microscope and a wound is made with a needle. The first change noticed in the surrounding tissue of the injured membrane is a dilatation of the vessels first of the arteries and then of the veins. In the arteries, there is a very noticeable acceleration of the flow of blood. At this early period, there is very little evidence of dilatation of the capillaries. In the course of an hour, however, expansion can be readily observed and the former invisible capillaries now fill with blood and are quite easily observed. In the course of an hour or two there is a slowing of the blood current. Before the wound was made, there was a well-marked central stream of corpuscles with an outer zone of plasma devoid of corpuscles. Now the central stream of corpuscles broadens out and the center zone of plasma becomes smaller and smaller. As it narrows, there is an increasing number of clear round blood leucocytes observed traveling at a slower rate than the central stream and occasionally stopping beside the walls of the vessels and after a short detention continue their course. The leucocytes act as though they wish to attach themselves to the walls of the vessels. The current becomes slower and slower until there is a vast distinction between the central and the peripheral streams. The corpuscles are now closely packed together and fill the whole surface of the blood vessel. The leucocytes now approach the vessel walls in large quantities and adhere more firmly. While the current is recognizable the action of the stream causes the leucocytes to assume a pear-shaped appearance the larger round end pointing in the direction of the current. As the blood stream gradually slows the corpuscles may at last move in a series of movements with the beats of the heart, while frequently in the veins and capillaries the mass of blood may be seen moving in one direction or the other. Frequently one or the other of these stages is followed by complete stasis of blood in the vessels of the injured area for occasionally little or no arrest is seen in the vessels. Accompanying this stage, there is already considerable exudation of clear fluid from the wound; there is an out-pouring of lymph from the distended vessels. With the slowing of the stream the leucocytes collect next to the walls of the small veins

and within the capillaries and pass from the interior to the exterior of the vessels. (Fig. 29.) These leucocytes after passing through the walls of the vessels collect in the lymph spaces between the vessel walls. There may also be found a small number of red corpuscles distributed among them. The leucocytes do not stop near the vessels but by an active amœboid movement they pass on to the point of injury. Then by the end of about six hours the surrounding area of the injury may be covered by a serum filled with leucocytes. Here then we trace the first step towards the provisional protection of the wound.

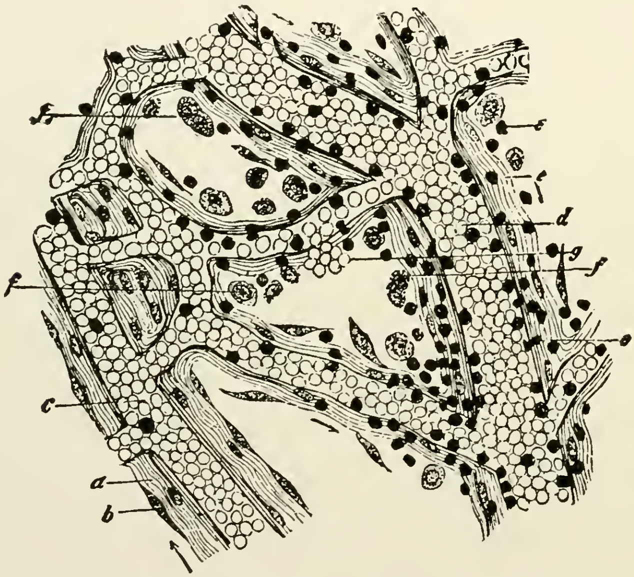


Fig. 29.—Inflamed Human Mesentery (Osmic-acid Preparation); *a*, Normal Trabecula; *b*, Normal Epithelium (Endothelium); *c*, Small Artery; *d*, Vein with Leucocytes Arranged Peripherally; *e*, White Blood-Cells, Which Have Emigrated or are Emigrating; *f*, Desquamating Endothelium; *f*, Multinuclear Cells; *g*, Extravasated Red Blood-Cells. x180. (Ziegler.)

If, in producing this wound irritating substances have not entered, the process may be arrested at this point. The stasis of blood in the distended vessels may now be followed by a restoration of the current and slow return of the vessels to their former caliber will take place.

On the other hand, if irritants of a microbic nature enter the wound the process may extend to abscess. If the irritants, in-

fections or poisons are too abundant, migration of quantities of leucocytes takes place and they collect between the bundles of connective tissue fibers. The cocci collect in the lymph spaces and the massing of leucocytes corresponds to the accumulation of the microbes. At the end of about forty-eight hours a complete abscess forms, separated sharply from the surrounding healthy tissue. In the center all traces of previous blood vessels are lost, while in the periphery they are easily traceable; in the center of the abscess the original tissue has wholly disappeared, while near the outer surface, sheaths and bundles of disintegrating fibers are seen; about the tenth day new growth of tissue begins to show itself. Numerous capillaries and newly-formed connective tissue are seen and the process of restoration takes place.

We have shown, in a general way, the different stages of inflammation in soft tissues of all vertebrates, including the gums and peridental membrane in man. The inflammatory process which takes place in the alveolar process, however, is quite different in its procedure. Instead of the traumatic injury to the frog just cited, the injury to the alveolar process is brought about by the poisons and toxins circulating in the blood and which I have called constitutional causes. These poisons and toxins take the form of local disturbance, of innervation, poor circulation and tissue metabolism. Generally the action of poisons is to contaminate all the blood which circulates to the remote organs. This general action of the poisons and toxins in the blood is added to the local action of irritants. These poisons act upon the nerve sheaths, decompose the blood but mainly affect the vessel walls. They also tend to accumulate in certain organs within which they frequently develop their chief action and often cause remarkable tissue change.

The principal structures affected by these poisons are the end organs, namely, the eye, the brain, the kidney recognized by every physician, while the dental pulp and alveolar process, which I have previously mentioned, are the most complete end organs in the human body and usually first involved. Because of the nature of their structures and surroundings, symptoms of disease are observed in these structures long before the other end organs are affected.

The dental pulp (as before stated) is the most complete end organ for the reason that the blood enters the tooth by a single artery. It then multiplies until the pulp is composed of myriads of minute arteries confined within bony walls. The return blood again passes through the apical end of the tooth by a single vein. It is not uncommon, therefore, for the arteries, filled with poisons, to expand and prevent the return to circulation, thus causing numerous diseases and frequently spontaneous death of the pulp.

The second most complete end organ is the alveolar process. It is the only bony end organ in the human body. The arteries

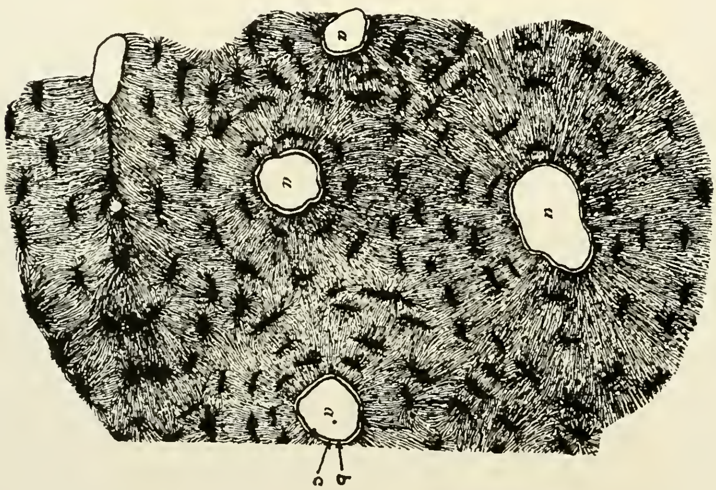


FIG. 30.—IS SIMILAR TO FIG. 10. THE HAVERSIAN CANAL SHOWS A DARK LINE AROUND THE INNER BORDER REPRESENTING THE ARTERIAL WALL.—THE SPACE BETWEEN THE DARK LINE AND THE BONE IS FILLED WITH FIBROUS TISSUE.

penetrate the bone in a wavy manner as far as the root of the tooth, which, so far as the disease is concerned, is a foreign body, when circulation ceases to any great extent.

The simplest illustration of the action of these poisons is that of a healthy individual at the sixth period of stress or about sixty years of age. This senile period, however, may take place at a much earlier time in life. The excretory organs become weakened and the poisons formerly excreted by the lungs, kidneys, bowels and skin are not eliminated but circulate in the blood. Odor from the breath, skin fæces, an abnormal urinary

acidity degree, indican and other poisons in the urine indicate these changes. These poisons are retained in the peripheral organs but more especially in the dental pulp and alveolar process. Those receiving the greatest quantity and retaining it are the ones so constructed as to prevent a return of the irritants or poisons to the circulation.

The irritants cause a dilatation of the vessels, first of the arteries and then of the veins. This dilatation of the arteries presses against the bony walls of the alveolar process which sets up absorption. (Fig. 30.) Since the poison is a general one

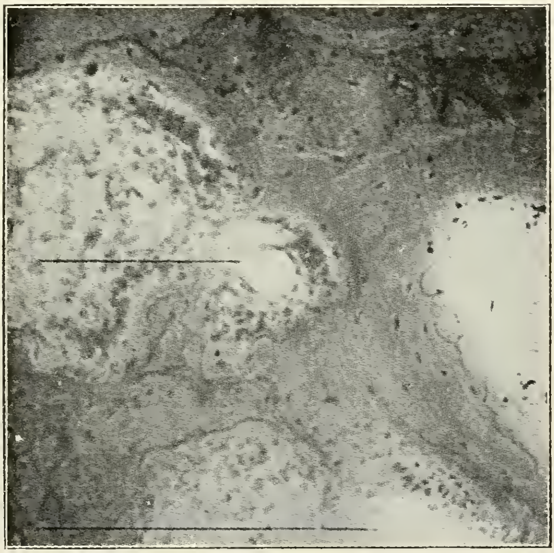


FIG. 31.—BONE ABSORPTION BY HALISTERESIS: IN THE MIDDLE SPACE THE BONE IS JUST BEGINNING TO ABSORB AROUND THE ARTERY.—THE SPACE TO THE LEFT SHOWS A LARGE PORTION OF BONE ABSORBED, LEAVING THE FIBROUS TISSUE IN PLACE; WHILE THE TWO SPACES AT THE RIGHT AND BOTTOM OF THE ILLUSTRATION SHOW THE LIME SALTS AND FIBROUS TISSUE ENTIRELY DESTROYED.

many arteries act in like manner. Chemical changes also take place around the arteries which assist greatly in producing bone absorption. Halisteresis is produced and the bone quickly disappears. Sometimes, especially when only a small area is involved lacunar absorption only occurs. The vessels of Von Ebner (capillaries) being much smaller, the irritation is not so intense nor is the effect of pressure or chemical changes upon the surrounding bone so great. The area of absorption, there-

fore, is limited, resulting in small canals from one Haversian canal to another or from one absorbed area by halisteresis to another. The tissue around the artery now is not unlike the soft tissue previously under consideration and the natural processes of inflammation continue. In many patients the process of inflammation stops with the absorption of bone. The tooth or teeth now are simply attached to normal fibrous tissue. In many, the inflammatory process proceeds a little further. There is now acceleration of the blood stream which later slows considerably. The central stream of corpuscles broadens and the outer zone of plasma grows smaller and smaller. The same process takes place in reference to leucocytes, red blood corpuscles and exudate as before (Fig. 31) and if there is sufficient irritation and infection, an abscess will form in the matrix or fibrous tissue which originally contained bony tissue. It will be seen, therefore, that the rigid bony framework of tissue prevents great vascular dilatation without bone absorption. It may, however, be the seat of great pain, due to pressure of the confined exudate upon nerve endings.

Local changes in function such as want of proper articulation; too great pressure in mastication on one or more teeth; slight change in position of teeth after they have once become solid in the jaw; destroying pulps and filling roots so that the peridental membrane is required to do more work; hypertrophies of roots; teeth that have once been abscessed and were apparently restored to health, etc., are a few of the local causes which earlier set up inflammation in the alveolar process by the irritants and poisons in the blood stream than in the normal adjoining teeth.

Local irritations such as wedging of teeth for filling; correcting irregularities of the teeth, etc., which have already set up local inflammation are fruitful sources and weak localities for the irritations in the blood stream to quickly renew inflammation and destruction of the alveolar process.

One of the best illustrations of the effect of poisons in the blood acting upon weak abnormal structures in the jaw is that of phosphorus poisoning. It is known that persons working in phosphorus, such as match-making, are liable to phosphor ne-

crosis of the jaws. When it occurs, it is usually associated with a carious or diseased tooth. It is supposed that the phosphorus enters the jaw through or around the tooth. This, however, is not the case. The poisons circulating in the air are taken into the system through the lungs or if handled by the hands through the skin. It thus enters the blood and causes a general disturbance of nutrition. The blood vessels become charged with the poison and the taking in of oxygen is considerably diminished. In consequence, degeneration of tissues results. The red blood corpuscles change their color and break down. The nervous system, especially the peripheral nerves degenerate. The arteries in the alveolar process are the first to become diseased. The parts of the jaw which first feel the effect of the poison are those parts where the teeth have lost their normal function, more particularly teeth with dead pulps whose roots may or may not be filled, with or without slight inflammation, teeth which have abscessed or been wedged or are doing more work than normal.

When absorption has taken place, the bone is not restored after the person has obtained his growth, the transitory nature of the process and only two sets of teeth being natural to man. If the process be removed from any cause (even if the permanent teeth be still in the jaw) it is not restored.

The absorption of the alveolar process, under constitutional causes, usually begins at the gingival border because of its thinness of structure. The gum tissue does not change but follows the bone, in its absorption, until the tooth is exfoliated or until, under treatment, the alveolar process is restored to health, when the gum attaches itself firmly to the bone and is restored to its normal condition.

We have shown the simple process of inflammation to the formation of abscess and the absorption of bone in relation thereto in the milder forms due to traumatism, toxins and poisons, but the procedure under severe constitutional conditions is much more intense.

The transitoiy nature of the alveolar process, its sensitiveness as an end organ, when inflammation is once set up in the gums, alveolar process or peridental membrane, due to either local or constitutional causes, compels it to become chronic with

disastrous results which are difficult of treatment and of restoration to health, especially if the vitality of the patient is low. This inflammatory process is along the line of least resistance.

THE NERVOUS SYSTEM IN INFLAMMATION.

There is much discussion by certain writers in regard to the action the nervous system plays in inflammation. Some take the stand that the central nervous system does act upon the blood vessels in a given locality while others claim that it must be a peripheral nervous mechanism which controls the blood vessels. Experiments have shown that the vascular changes connected with inflammation can occur independently of the central nervous system. It follows then that there may be a peripheral nervous mechanism controlling the vessels.

It has been demonstrated by Klebs [1] that the endothelial walls of the capillaries do contract. The conclusion then is that the endothelium of the capillaries is, to some extent, self regulating. It is quite possible that the muscular coats of the smaller arteries and capillaries act to stimuli.

Thoma [2] says, "It is evident, however, that the local circulatory reaction after injury depends upon the condition of the cerebro-spinal vasomotor centers and that the peripheral vasomotor nerve apparatus may also act independently. From what has been said it appears that among the direct action of traumatism, injury to the nerves is of great importance. This injury to the nerves at the site of the lesion is either a change in the invisible molecular structure of the nerve fibers, or one which can be recognized under the microscope. Each change in the molecular structure of the nerve causes a change in its excitability which is termed 'nerve irritation' by physiologists. This irritation is frequently more or less painful, and is perceived subjectively as pain. The disturbance of the innervation of the vessel wall, however, is to be clearly distinguished from the sensation of pain. The former also is partly caused by the direct action of the injury to the vasomotor nerve apparatus contained

[1] Klebs. Allg. Pathologie.

[2] Thoma. Pathology and Pathological Anatomy.

in the injured area. The disturbance of vasomotor innervation, however, is seldom strictly limited to the area directly affected by the injury, since the nerve irritation both directly and reflexly alters the excitability of the vasomotor centers in the walls of the neighboring arteries, and thus causes the local circulatory reaction.''

My researches have shown that the nerve supply in the alveolar process, as compared with that in the peridental membrane and periosteum is very slight. Inflammation and infection, therefore, taking place in the peridental membrane at the apical end of the tooth root forming alveolar abscess or at the side nearest to the apical end where peridental abscess forms, where the alveolar wall is quite thick, pain is much more severe than when the abscess forms in or near the gingival margin of the process. On account of local and constitutional irritations, nerve end degeneration in alveolar absorption is the rule. Pain, however, in this instance is not as severe in abscess formation in the alveolar process as in other bones.

These local and constitutional irritations produce paralysis and finally death of the nerve fiber. When degeneration or death of the nerve fiber takes place, the action upon the coats of the arteries is such that inflammation and absorption of the alveolar process ensues.

The local irritations and infections produce the same effect upon tissue in inflammation as the internal (constitutional) irritations and infections. Adami [3] says, ''Anything which causes local injury to the tissue is a cause of inflammation, be it a mechanical trauma, a physical insult, as by heat, cold or electricity, a disturbance brought about by altered metabolism and abnormal internal secretions, or bacterial or microbic invasion and growth. This last is the commonest cause of acute reaction and differs from the physical and mechanical causes (although not from metabolic disturbances) in that, as a cause, it is not of momentary duration, but of continued. It is not the mere physical entry of microbes into the tissues that induces inflammation but the liberation of them of their products in growth or disintegration. And so long as those products are being liberated,

[3] Adami. Principles of Pathology, Vol. 1, page 420.

for so long is the cause of action. It differs from the metabolic causes in that the latter induce tissue irritation of a milder grade and do not induce acute, but rather chronic reaction."

Kirk[4] says, "Viewed as an inflammatory process we have, then, in the study of pyorrhœa (interstitial gingivitis) to regard its clinical or objective phenomena as reactions of the retentive tissues of the teeth toward injuries inflicted by mechanical trauma, physical irritants, altered metabolism, the toxic effects of altered secretions, or by the toxic products of microbic or bacterial invasion. Any of these agencies, severally or collectively, may induce such changes in the retentive structures as will lead to their molecular necrosis and the ultimate exfoliation of the teeth, the process constituting comprehensively what we know as pyorrhœa alveolaris (interstitial gingivitis)."

The process of inflammation from local irritation and infection proceeds in the same manner as the traumatism upon the web of the frog's foot.

Patients who have been ill for any length of time, such as those suffering with phthisis, lues, kidney lesions or other lingering diseases, and those with low vitality, take on interstitial gingivitis much more readily than those of strong vitality.

Summing up this chapter, no matter whether the irritation be local or constitutional or both, the inflammation set up thereby is progressive in its nature and does not cease until the tooth or teeth have been exfoliated by the absorption of the alveolar process, although treatment, changes in environment and systemic conditions may check the disease for a limited time. The inflammation is solely the cause of bone absorption and may be slow or rapid in its action.

---

[4] American Text-Book of Operative Dentistry, Fourth Edition, page 470.

CHAPTER XIV.

RESEARCHES ON ANIMALS IN INTERSTITIAL GINGIVITIS.

All vertebrates possessing two sets of teeth during life the roots of which are situated in bony sockets have interstitial gingivitis to a greater or lesser extent. The mere fact of the eruption and shedding of the teeth, as in human, is an indication that there is an inflammatory process present. Wild animals leading a natural, normal life are less liable to have this disease than those having become domesticated. Wild animals domesticated and tamed animals taken out of their natural environment suffer with this disease to as marked a degrees as human. While it is true animals, as a rule, do not suffer with the nervous types of disease like human, yet a wild animal in confinement with artificial feeding must suffer, to a greater or lesser extent, according to the nature of the change and the amount of restlessness produced. Changes in vital resistance are not as frequent or as marked in animals as in human, yet wild as well as domesticated animals do occasionally suffer with disease and changes in vital resistance.

Wild animals roaming at large and obtaining their food are in a much more natural environment than when in captivity. The organs of the body are performing their natural functions, and thus elimination is carried on normally. Old age, even in animals living in their natural wild environment, predisposes weakened eliminating organs, while these, in turn, owing to poisons in the blood, will cause interstitial gingivitis and finally loss of teeth.

Wild animals under domestication owing to change of environment can best be studied in relation to this disease. A visit to the great Zoological Gardens in Dublin, London, Hamburg, Paris, New York City or Chicago, and a careful study of the mouths and jaws of the animals there confined will readily convince the investigator of the truth of these statements. Monkeys are probably more susceptible to disease and death

than most animals. Cows fed upon brewers' slop and confined indoors have interstitial gingivitis badly; horses which have been biting grass with their front teeth, living in the sunshine and breathing the fresh air, when returned to the stable in the Fall begin to "crib," biting the woodwork around the stall. The front teeth feel uncomfortable because of the entire change of environment. The "cribbing" causes the blood to be forced out of the capillaries of the peridental membrane, and for the time being gives relief to the animal. It soon returns and because of the pain the process is repeated. In the course of a week or two, however, the eliminating organs adjust themselves to the new environment and the teeth remain comfortable. House animals are very prone to interstitial gingivitis as well as to other constitutional diseases. In most animals of this kind they are taken out of their natural environment, and autointoxication and disease is the rule. The carnivora suffer with pyorrhœa alveolaris to a more marked extent than the herbivora owing to the fact that they subsist to a great degree upon pus-ladened nitrogenous food, thus infecting the gums and peridental membrane. Dogs with this disease are the best animals for research.

As the first step in investigation, two practitioners of comparative medicine, with an extensive hospital practice (Dr. Charles E. Sayre and Dr. Alsop E. Flower), were consulted as to the frequency of this disease in animals. All animals under their care suffered from it more or less, but eighty per cent of dogs over eight years of age had the disease. Nearly every dog in the hospital under their care was so affected. These dogs comprised all breeds, from spaniels and terriers to the Newfoundland, St. Bernard and great Dane. On examination, every phase of interstitial gingivitis was found in the mouths of these dogs, from its inception to the loss of the teeth. The number of dogs observed was twenty-seven. The roots of the teeth of some were covered with deposits and so exposed that the teeth could be removed with the fingers. Such diseased mouths are rarely, if ever, present in human beings. The outer plate of bone was absorbed, the roots entirely exposed, pus was oozing from around them and the mucous membrane was badly inflamed.

It should be remembered that the jaw of the dog, like the jaw of man, is undergoing considerable variation. Like man, the dog, having put himself under new social conditions (so to speak), is varying greatly both as to brain, skull and jaw from his wolf-like ancestor. As he is under the protection of man, the struggle for existence as to food is less intense than

FIG. 32.—THE MOUTH OF A SCOTCH TERRIER, SOME OF THE TEETH HAVE BEEN REMOVED ON ACCOUNT OF INTERSTITIAL GINGIVITIS.

in the wild state and consequently there is less occasion, even for fighting purposes, of his jaws and teeth.

Independently of conditions of this type, many of the dogs suffered from constitutional disorders. Eight had skin diseases which in the dog are more likely to produce obvious con-

stitutional defects than in man. Some were old and blind. Some had been injured and were under treatment for wounds. Some were suffering from rachitis, nervous diseases, and were overbred. Others were constipated or had germ types of diarrhœa. One had kidney inflammation and bronchitis with high

Fig. 33.—The Mouth of a Boston Terrier; Owing to Illness Interstitial Gingivitis has Caused Absorption of Bone and the Incisors and Molar were Removed by the Fingers.

fever. In short, these dogs, being house dogs, presented most of the constitutional diseases to which man is liable.

The mouth of a Scotch terrier is shown in Fig. 32. The molar and premolar had been removed with the fingers. The cuspids and incisors are quite loose. There are large deposits

of tartar. The gum and alveolar process have been absorbed nearly one-half the length of the roots of the teeth. In Fig. 33 is seen the mouth of a Boston terrier with the incisors and premolars removed. There is extensive pyorrhœa. There are calcic deposits upon the cuspids and molars. There is recession of the gums and alveolar process. In Fig. 34 is shown the mouth

Fig. 34.—The Mouth of Another Boston Terrier; One Tooth has been Removed and Interstitial Gingivitis is Seen Around the Other Teeth.

of another Boston terrier. In it one premolar in the upper and one on the lower jaw have been extracted. There is extensive inflammation of the gum about the molar, cuspid and incisor with large calcic deposits about the teeth. In Fig. 35 are shown teeth covered with calcic deposit the entire length of the root.

These teeth were removed by the fingers from the mouths of two dogs, one of which was later obtained for scientific study. This was all the material to be obtained from the hospital, since the dogs were pets which had been placed under treatment by their owners.

Through the courtesy of Poundmaster Hugh Curran, the necessary material was obtained from the Chicago Dog Pound. Here from four hundred to a thousand dogs are killed per week during June, July and August each year. Ninety-five per cent are mongrel curs leading a street life, hence neither luxurious diet nor luxurious care can be charged with any disease in them. They have, at least, plenty of outdoor exercise and fresh air. Many, despite this reversion to the life of their wolflike ancestors, have skin diseases and are deaf and blind from old age. The bodies were secured after death, at which time exam-

Fig. 35.—Shows Teeth of a Dog Covered with Calcic Deposits which were Quite Loose and were Removed by the Fingers.

inations of the mouths were made. Five per cent of the dogs entering the pound are of good breeds. These, if not called for by the owners, are sold for a moderate price.

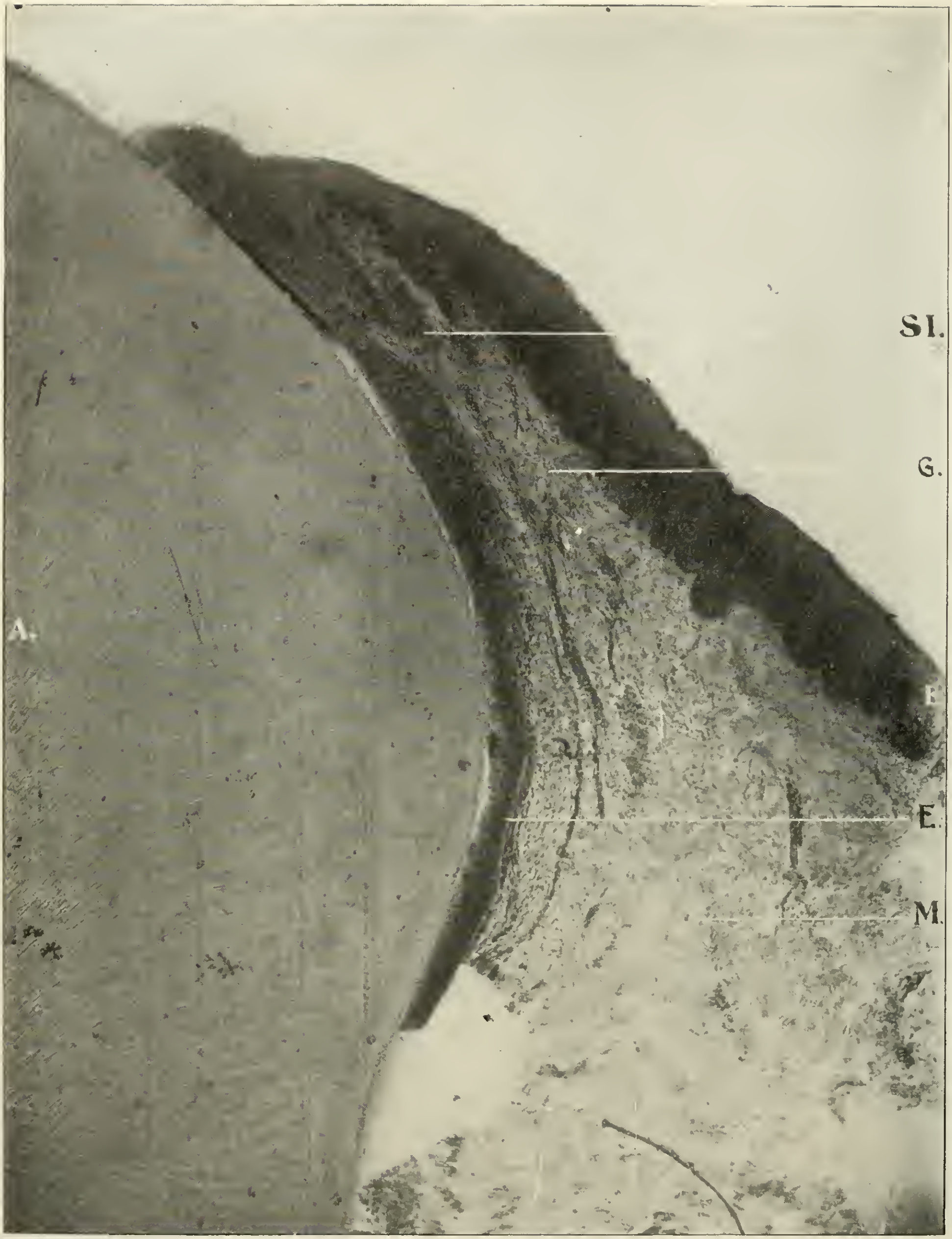
The dogs selected for the death penalty are collected in a large box pen, leading out of which is a door through which they pass into an air-tight box. Communicating with this box is a stove in which sulphur is burned with charcoal. The fumes pass into the box and death is almost instantaneous and painless. After they remain fifteen minutes, a door leading to the air is opened and the bodies are carted away. It was at this time that access was had to them. The mouths were then examined. Such cases as were of interest were placed on one side and the jaws removed from the bodies. Inside of one-half hour the specimens were in a solution to be kept until desired for use. Jaws (with interstitial gingivitis in all stages of progress, from simple in-

flammation of the gums to the most extreme cases of exfoliation of the teeth) were obtained in an abundance for future studies. It is not an easy matter to ascertain the ages of these animals. In a general way, it was found that inflammation of the gums, especially about the canine teeth, was almost always present in dogs over one year. About twenty-five per cent of these dogs at four years of age had the disease, eighty per cent at from eight to ten years, ninety-five per cent over twelve years of age. Since I commenced my investigation (twenty years ago), I have examined quite a large number of dogs about homes, but have never found a dog over four years without this disease to a greater or less extent. Many house dogs at one year had inflammation of the gums. Dogs for infection and those used for mercurialization were picked up in the streets.

Most of the dogs exhibited at dog shows are young, ranging from one to four years of age. About twenty-five per cent would range four years to eight. A casual examination of their mouths revealed interstitial gingivitis. Occasionally recession of the gums and pyorrhœa alveolaris occurred. On a more careful examination, twenty-five per cent of dogs between the ages of one and four were found to have interstitial gingivitis and seventy-five per cent of dogs from four to eight years were found to have interstitial gingivitis with recession of the gums and pyorrhœa alveolaris. In the study of this disease, therefore, dogs are excellent substitutes, since for pathologic research they can be obtained at any stage of the disease.

The technique of the examinations of interstitial gingivitis and pyorrhœa alveolaris in dogs was as follows: After fixing and hardening in two per cent formalin, alcohol, or Muller's fluid, the tissues were decalcified in a five per cent alcoholic solution of nitric acid, imbedded in celluloidin and stained in various ways, the principal ones being hæmatoxylin and eosin. Ten or more slides would be obtained from each tooth. Out of these slides have been selected a series illustrating the progress of the disease from beginning to the loosening of the tooth.

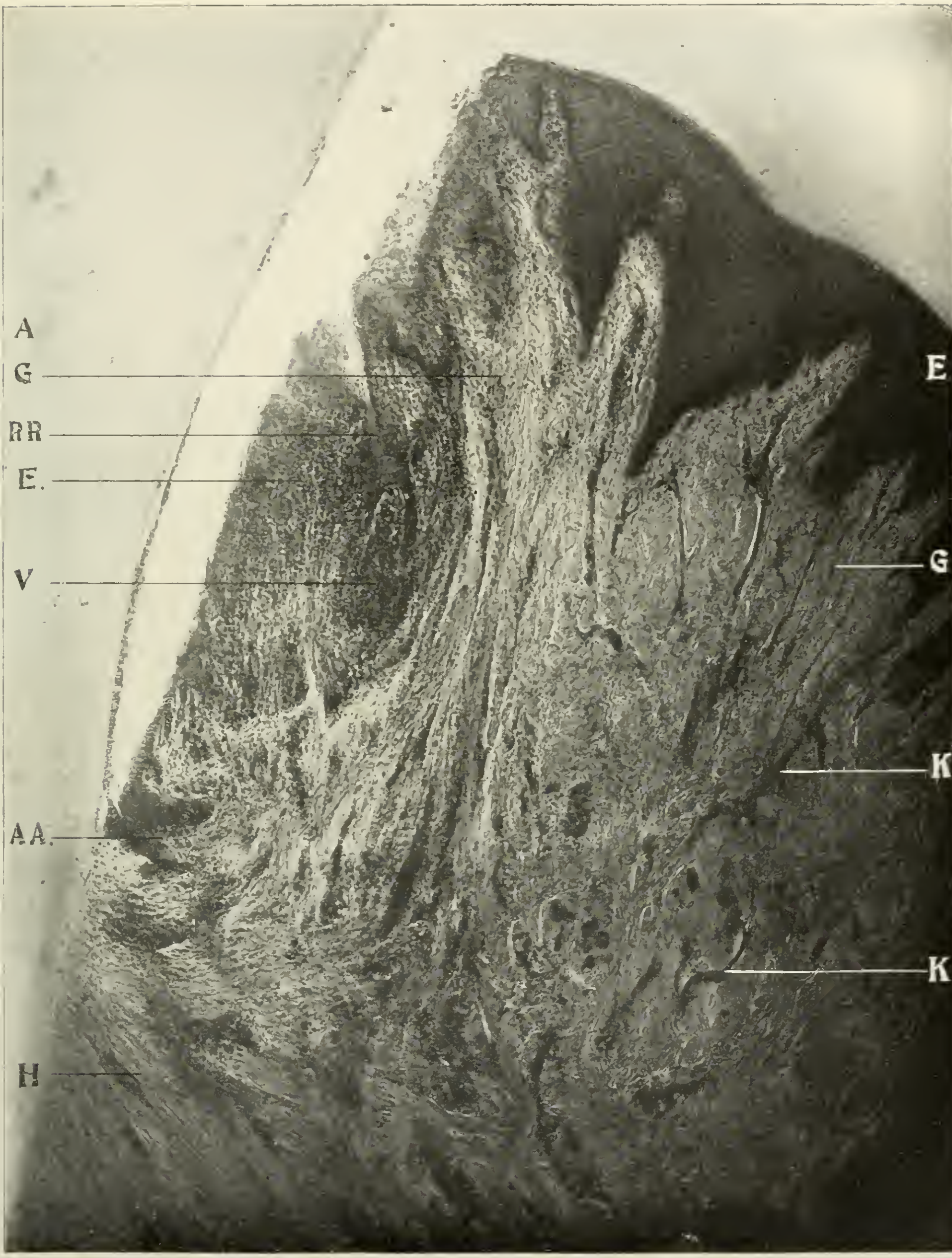
Fig. 36 is a longitudinal section of a cuspid tooth with the alveolar process in situ. A illustrates the enamel of the tooth, (E) the epithelium passes from the outer margin to the lower



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

FIG. 36.—LONGITUDINAL SECTION OF TOOTH AND GUM TISSUE. SLIGHT GINGIVITIS. DOG.

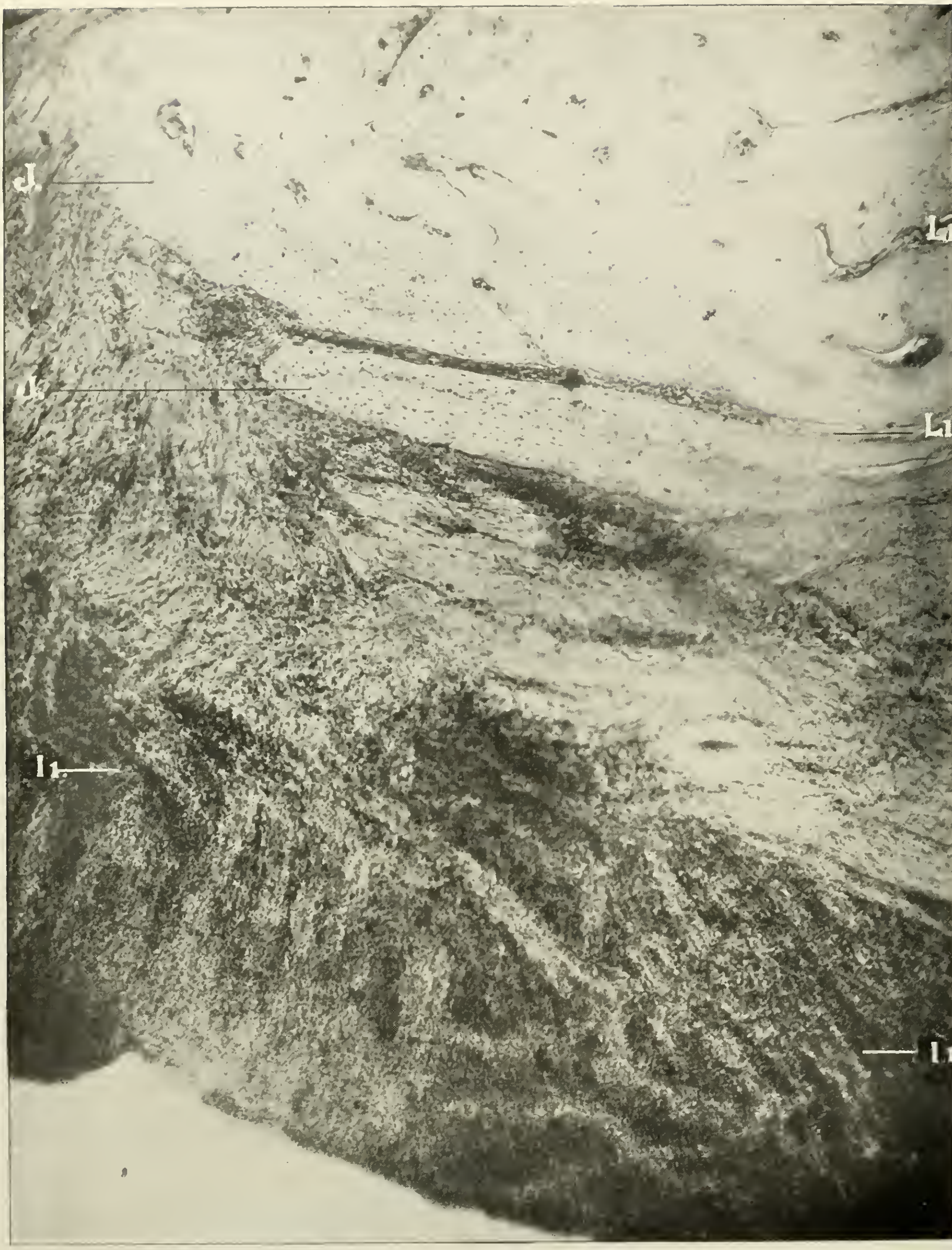
A, Enamel. E, Epithelial Tissue. G, Submucous Membrane. M, Fibrous Tissue.
SI, Slight Inflammation.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

FIG. 37.—LONGITUDINAL SECTION OF TOOTH AND GUM TISSUE. CHRONIC INTERSTITIAL GINGIVITIS. DOG.

A, Enamel. E, Epithelial Tissue. G, Submucous Membrane. H, Periosteum. K, Capillaries. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. RR, Space Pocket from want of Union of Epithelial Fold.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 38.—Longitudinal Section of Alveolar Process and Peridental Membrane. Slight Interstitial Gingivitis, Extending into Alveolar Process. Dog.

J, Alveolar Process. L^1, Inflammation Extending through Enlarged Haversian Canals. I^1, Inflamed Peridental Membrane.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 39.—Longitudinal Section of Alveolar Process and Peridental Membrane. Chronic Interstitial Gingivitis, Extending into Alveolar Process. Dog.

H, Periosteum. J, Alveolar Process. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. I^1, Inflamed Peridental Membrane. L^1, Inflammation Extending through Enlarged Haversian Canals.

border, then folds upon itself and extends down the side of the crown of the tooth as far as the neck. Unfortunately, in this specimen, the structure connecting the epithelium and the fibrous tissue of the periosteum has been destroyed. The papillary layer of the sub-epithelial tissue is plainly shown at the outer border. Small round-cell inflammation may be seen extending along the border of this layer. It can also be observed extending down the capillary blood vessels into the submucous tissue (SI and G).

Fig. 37 shows a similar section of another tooth. Here the epithelial structure (E) is pulled away slightly from the edge of enamel (A). In this section the infolding of the epithelium is shown at the neck of the tooth. This structure passes downward, folds outward and upon itself (AA) and returns two-thirds of the distance toward the gingival border, leaving a pocket (RR). The epithelium (E) is very dense and thick. The papillary layer of the submucous tissue (G) is very clearly defined. The capillaries (K) can be distinctly traced from the deeper fibrous tissue through the submucous layer into the papillary layer. The thick and heavy fibrous tissue of the periosteum ("Dental Ligament," Black) may be seen at H, inserted firmly into the cementum and extending outward and downward. Just below (AA) may be seen the interlacing of the coarser fibers of the periosteum with the finer fibers of the submucous tissue. Chronic round-cell inflammation may be seen extending from the papillary layer through the capillaries into the interstitial tissue of the submucous layer and the periosteum. Marked inflammation has occurred at V. The openings in the folds of the epithelium are fruitful sources for the accumulation of food, epithelial scales and detritus, in which fermentation and decomposition from micro-organisms result, producing inflammation.

Fig. 38 is a section through the peridental membrane (I) and alveolar process (J) at the lateral incisor. The inflammation has extended down from the papillary layer through the submucous tissue, the fibrous tissue of the periosteum into the peridental membrane and into the alveolar process. Round-cell inflammation may be seen in the blood vessels extending through the Haversian canals (L^1).

Fig. 39 is a similar section from another tooth showing

chronic inflammation extending throughout the peridental membrane (I) and alveolar process (J). The Haversian canals (L) are well outlined by the inflammatory progress. Marked inflammation has resulted at V and also at the margin of the alveolar process.

Fig. 40 is a section of the peridental membrane and alveolar process, illustrating the effect of interstitial inflammation upon the blood vessels and alveolar process. Chronic inflammation extends throughout the peridental membrane with very decided inflammatory change (V). The cut ends of the blood vessels which were originally situated in the Haversian canals are seen (BV). They have become involved with the result of a thickening of the walls and endarteritis obliterans. The bone about these vessels has been entirely absorbed. The inflammation has extended beyond, into and through the Haversian canals, producing the type of absorption of the trabeculæ known as halisteresis ossium. Lacunar absorption has also occurred (O). Where decided inflammation (V) has taken place, abscesses are more liable to occur (as will be noticed later) from the large number of blood vessels at this locality.

Fig. 41 is a section from another location of the alveolar process with a greater amplification, showing the inflammatory process extending through the alveolar process. Endarteritis obliterans may be seen in different localities (EO). Three forms of absorption are evident in this figure: Enlarged areas arising from absorption of the trabeculæ (halisteresis ossium) due to the inflammatory process. The vessels of Von Ebner precede perforating canal absorption (BB), distributed over the entire field, also the result of the inflammatory process and lacunar absorption (O) which may result from inflammation. As long as the fibrous tissue remains in these large areas to retain the osteoblasts, new bone tissue may be produced under favorable conditions. On the other hand, when this tissue and the osteoblasts are destroyed, the alveolar process cannot be restored.

Fig. 42 shows a section of the alveolar process from another dog. Here lacunar and other absorption (halisteresis ossium) are well shown. Thirty-seven osteoclasts (O) may be counted in the field while destruction of bone by halisteresis (Q) is rapidly

going on. Remains of Haversian canals with the blood vessels may be seen (BV, L). In the discussion of the peridental membrane extending into the alveolar process (page 37), particular attention was called to the fact that large bundles of fibers extended into the process in such a manner as almost to isolate portions of bone. In the lower left-hand corner (X) may be seen two pieces of the alveolar process entirely separated from each other and the main body of the bone. In interstitial gingivitis, it is not uncommon to find pieces of the alveolar process separated by halisteresis and lacunar absorption. When loose teeth are extracted as a result of this disease, pieces of the alveolar process come away with the peridental membrane attached to the tooth. Fig. 89 was obtained in this manner. In the upper left-hand corner may be seen eight or ten new osteoclasts (O) in an enlarged Haversian canal, at work isolating one piece of the alveolar process from the other.

Fig. 43 shows a slide from still another dog. Halisteresis (Q) and perforating canal (P) absorption are here well shown. In the larger space at the lower left-hand corner may be seen two arteries (EO) which were originally the location of Haversian canals and which have thickened walls and a tendency to obliteration. The light color shows decalcification, the dark normal bone. At P may be seen perforating canal absorption. At FG fat globules may be seen, while in the larger space at the upper right-hand corner is evident entire destruction of the fibrous tissue.

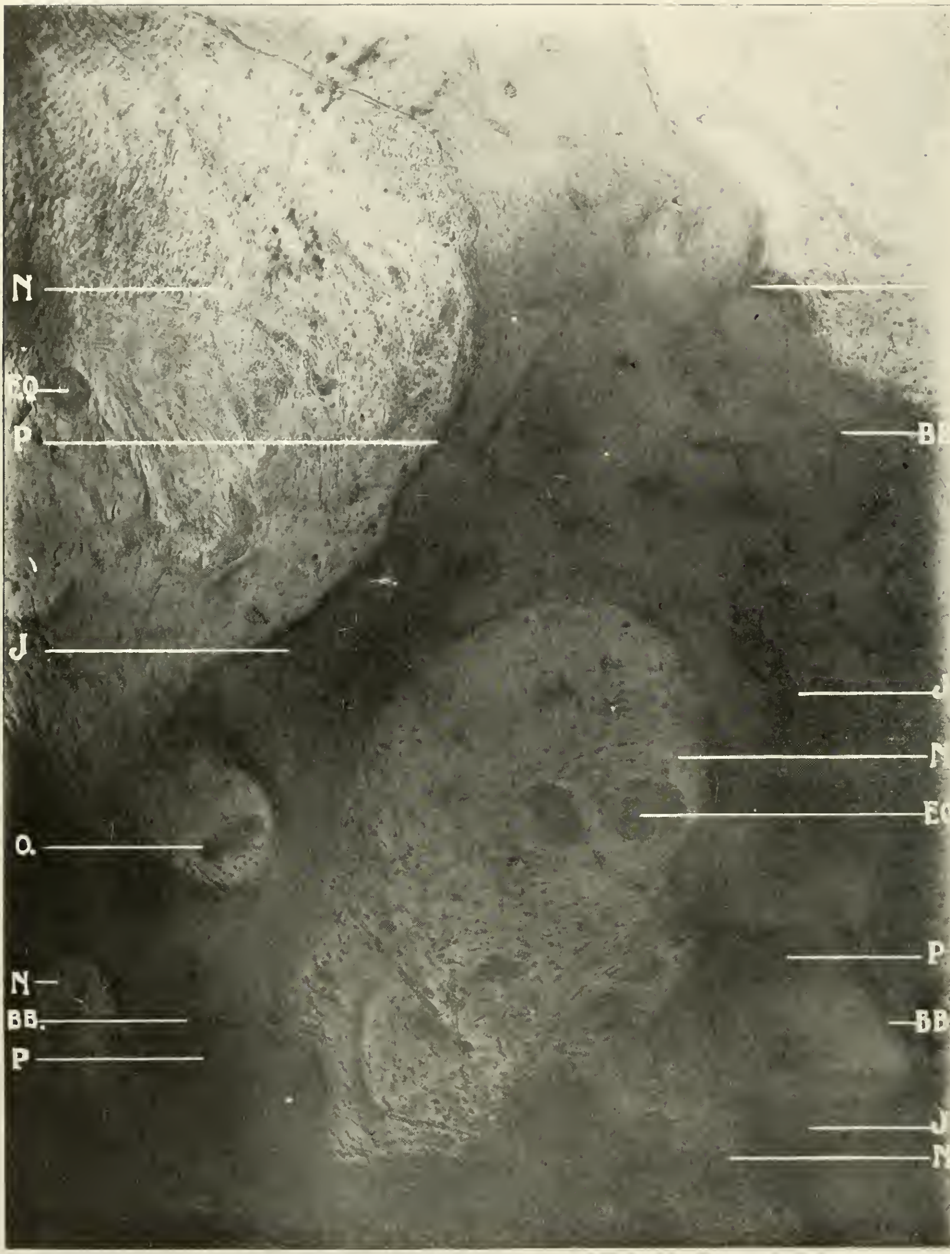
Fig. 44 illustrates a cross section of alveolar process and cuspid root, showing absorption of the root. Inflammation extends throughout the peridental membrane (I). The capillaries (K) are quite numerous. These are cut both crosswise and lengthwise. Absorption (S) of the root may be seen progressing at these localities.

Fig. 45 shows a longitudinal section of the end of the root. Active destruction has been going on both in the pulp chamber (D) and at the external surface of the cementum (C). The irritation and inflammation has caused the odontoblasts to fill up the pulp chamber with secondary dentine, and obliteration of the chamber has taken place. Below the constricted pulp may be

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 40.—Longitudinal Section of Tooth, Alveolar Process and Peridental Membrane. Violent Round-Cell Inflammation of Peridental Membrane, Extending through the Haversian Canals into the Alveolar Process.

C, Cementum. J, Alveolar Process. K, Capillaries. L, Haversian Canals. N, Large Spaces arising from Absorption of the Trabeculæ, starting in the Haversian Canals (Halisteresis). O, Lacunar Absorption. V, Violent Inflammation. BV, Blood Vessels, originally Haversian Canals. I[1], Inflamed Peridental Membrane. L[1], Inflammation Extending through Enlarged Haversian Canals.



X 150. D. D. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 41.—Longitudinal Section of Alveolar Process. Chronic Inflammation Extending throughout, showing Halisteresis, Perforating Canal and Lacunar Absorption. Dog.

J, Alveolar Process. N, Large Spaces arising from Absorption of the Trabeculæ, starting in the Haversian Canals (Halisteresis). O, Lacunar Absorption. P, Perforating Canal Absorption. BB, Blood Vessels of V. Ebner preceding Perforating Canals. EO, Endarteritis Obliterans.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

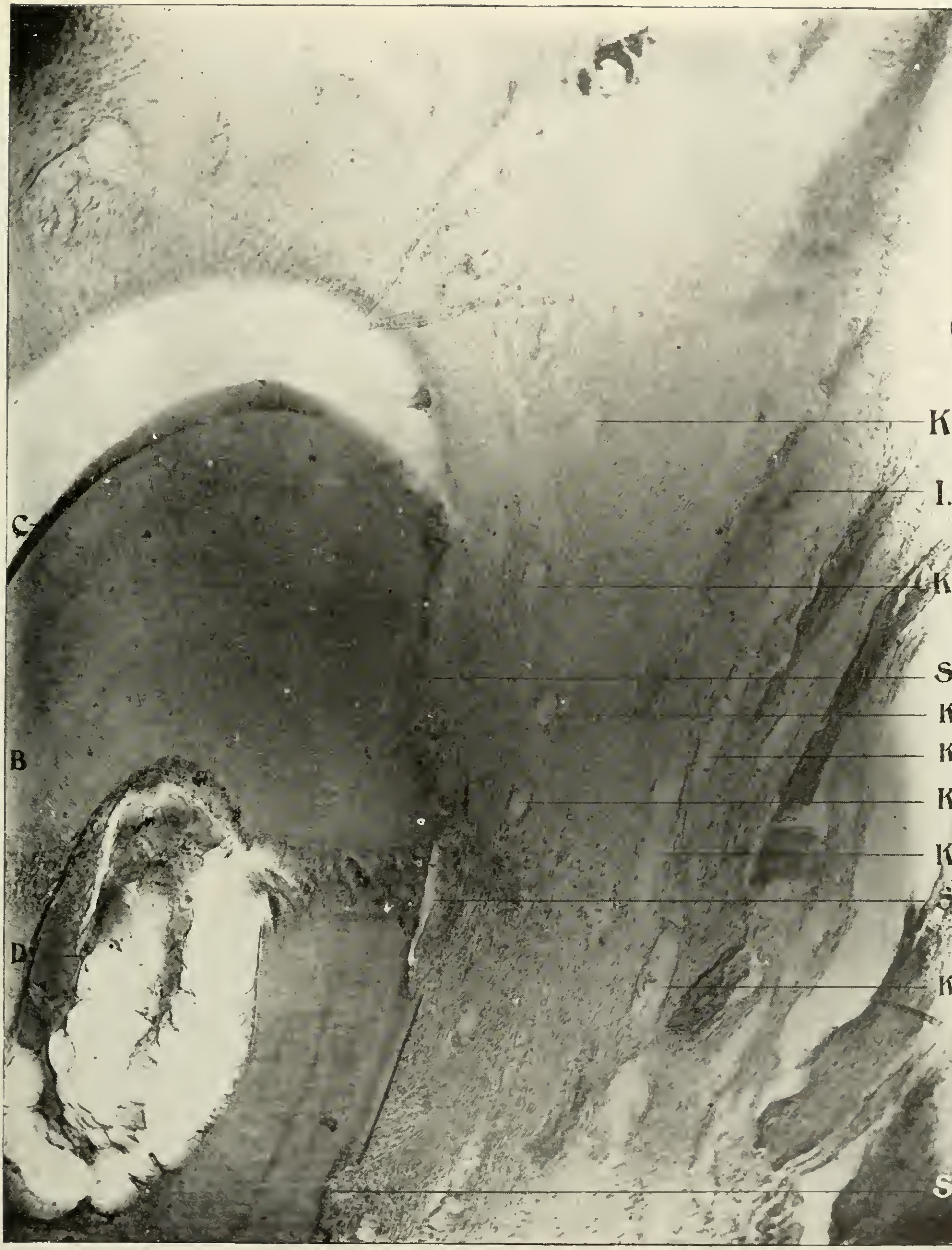
Fig. 42.—Longitudinal Section of Alveolar Process. Chronic Inflammation Extending throughout, showing Halisteresis and Lacunar Absorption. Dog.

J, Alveolar Process. L, Haversian Canals. N, Large Spaces arising from Absorption of the Trabeculæ, starting in the Haversian Canals. O, Lacunar Absorption. Q, Halisteresis Ossium or Decalcified Bone. X, Remains of Calcified Bone. BV, Blood Vessels originally Haversian Canals.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

FIG. 43.—TRANSVERSE SECTION, ALVEOLAR PROCESS. CHRONIC INFLAMMATION EXTENDING THROUGHOUT. DOG.

J, Alveolar Process. N, Large Spaces arising from Absorption of the Trabeculæ, starting in the Haversian Canals. P, Perforating Canal Absorption. Q, Halisteresis Ossium or Decalcified Bone. X, Remains of Calcified Bones. EO, Endarteritis Obliterans. FG, Fat Globules.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

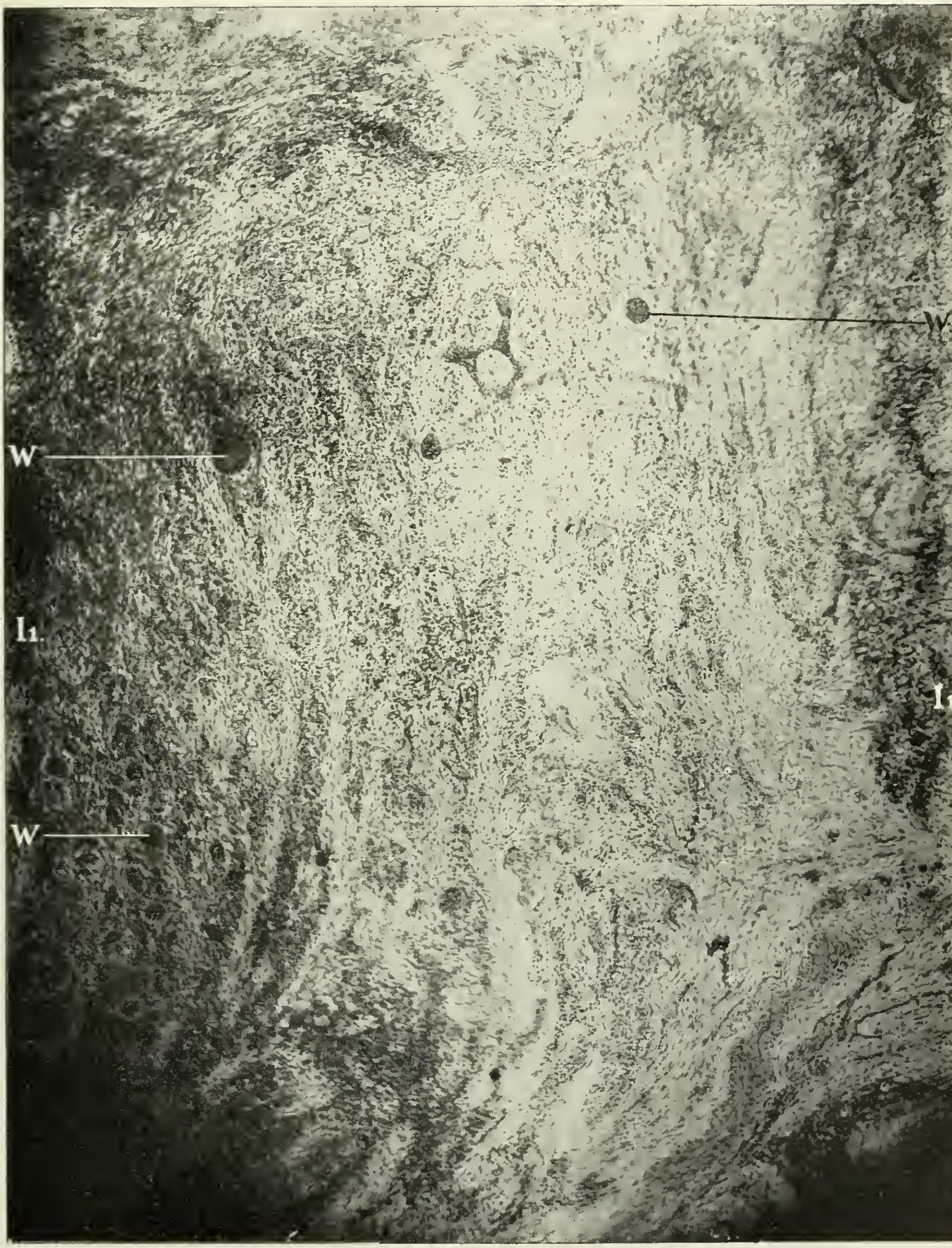
FIG. 44.—CROSS SECTION OF TOOTH, ALVEOLAR PROCESS AND PERIDENTAL MEMBRANE. CHRONIC INFLAMMATION OF PERIDENTAL MEMBRANE AND ABSORPTION OF THE ROOT OF TOOTH. DOG.

B, Dentine. C, Cementum. D, Pulp. I^1, Inflamed Peridental Membrane. K, Capillaries. S, Root-absorption.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 45.—Longitudinal Section of the End of the Root of a Tooth, Alveolar Process and Peridental Membrane, Showing Chronic Inflammation of the Peridental Membrane. Exostosis of the Root of the Tooth and Lacunar Absorption. Dog.

C, Cementum. D, Pulp, with 3 Foramina. J, Alveolar Process. O, Lacunar Absorption. P, Perforating Canal Absorption. CC, Cementosis.



X 75. A, A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 46.—Cross Section of Inflamed Peridental Membrane. Dog.

I1, Inflamed Peridental Membrane. W, Epithelial Débris.

X 15. 75 M. M. obj. Spencer. Micro-photograph, reduced one-third.

FIG. 47.—LONGITUDINAL SECTION OF TOOTH, ALVEOLAR PROCESS, PERIDENTAL MEMBRANE, SHOWING INTERSTITIAL GINGIVITIS AND PYORRHOEA ALVEOLARIS, WITH TOOTH ABOUT TO BE EXFOLIATED. DOG.

C, Cementum. E, Epithelial Tissue. H, Periosteum. I, Peridental Membrane. J, Alveolar Process. K, Capillaries. L, Haversian Canals. M, Fibrous Tissue. R, Pus Pockets. U, Nerve Tissue. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. CC, Cementosis. DD, Calcific Deposits Destroyed by Acids.

X 40. 35 M. M. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 48.—Longitudinal Section of Tooth, Alveolar Process, Peridental Membrane and Gum Tissue, Enlarged from Fig. 42, Showing Active Inflammation, with Pus Pocket. Dog.

C, Cementum. E, Epithelial Tissue. G, Submucous Membrane. I^1, Inflamed Peridental Membrane. J, Alveolar Process. L^1, Inflammation Extending through Enlarged Haversian Canals. M^1, Inflamed Fibrous Tissue. R, Pus Pocket. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. FF, Food Containing Micro-Organisms.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 49.—Longitudinal Section of Tooth, Alveolar Process, Peridental Membrane and Gum Tissue, Enlarged from Fig. 42, Showing Active Inflammation with Pus Pocket. Dog.

C, Cementum. E, Epithelial Tissue. J, Alveolar Process. M[1], Inflamed Fibrous Tissue. R, Pus Pocket. V, Violent Inflammation.

seen three divisions of the pulp (D) extending through three separate canals in the cementum (C). Cementosis (CC) may be seen at the end of the root. Lacunar absorption is going on (O). Thus results a building up and tearing down of the same tissue from the same cause, interstitial gingivitis.

Fig. 46 shows inflammation of the peridental membrane (I^1) with epithelial debris (W) scattered throughout the field.

Fig. 47 is a section through the jaw and incisor tooth, showing the relation of the structures to each other in a severe case of interstitial gingivitis and pyorrhœa alveolaris. The tooth is attached at only a very small portion of the apical end of the root. The disease has been of long standing. Absorption of the alveolar process on one side has progressed on fully one-half of the root, while upon the other about one-third the distance. Inflammation commenced at the gingival border and extended through the periosteum (H), peridental membrane (I) and alveolar process (J). Marked inflammation (V) has occurred in the mucous membrane fold. An abscess has formed with a fistula extending to the gingival border. The thin border at the left of the fistulous tract is the epithelium layer next to the tooth. It is evident that the pus burrowed to the surface through the structure instead of between the epithelium and the tooth. A similar abscess and fistulous tract are evident upon the gingival border on the opposite side of the tooth. The irritation produced by the movement of the tooth has caused the cementoblasts to deposit large quantities of material upon the sides and the end of the root. The main nerve trunks (U) may be seen at and below the end of the root.

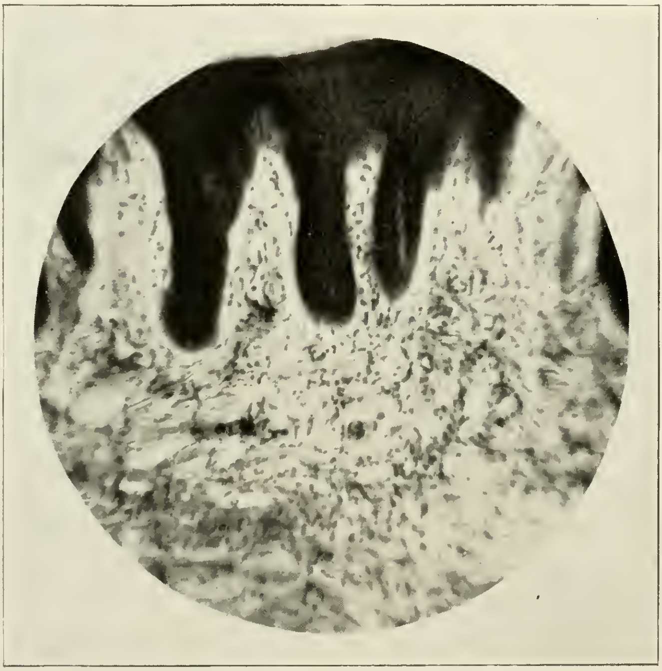
Fig. 48 illustrates the alveolar border on the right side of Fig. 45, greatly amplified. This shows the progress of interstitial gingivitis extending through the alveolar process producing absorption with intense inflammation of the peridental membrane and abscess with fistulous tract.

Fig. 49 shows a similar process amplified from the left side of Fig. 47. It is interesting to note in this illustration that the fibers of the sub-epithelium pass down and become interwoven with the coarser fibers of the periosteum in just the opposite direction from those in the other side of the tooth, and in other

illustrations. The fibers from the mucous membrane along the side of the tooth extend down and into the peridental membrane without a break in the structure. The arrangement of the fibers of the submucous layer in producing the fold is well illustrated in the figure. This picture illustrates inflammation starting in the gingival border.

MERCURIAL INTERSTITIAL GINGIVITIS IN DOGS.

To secure a chain of evidence that interstitial gingivitis (due to the metals, drugs, uric, lactic and other acids) commenced in



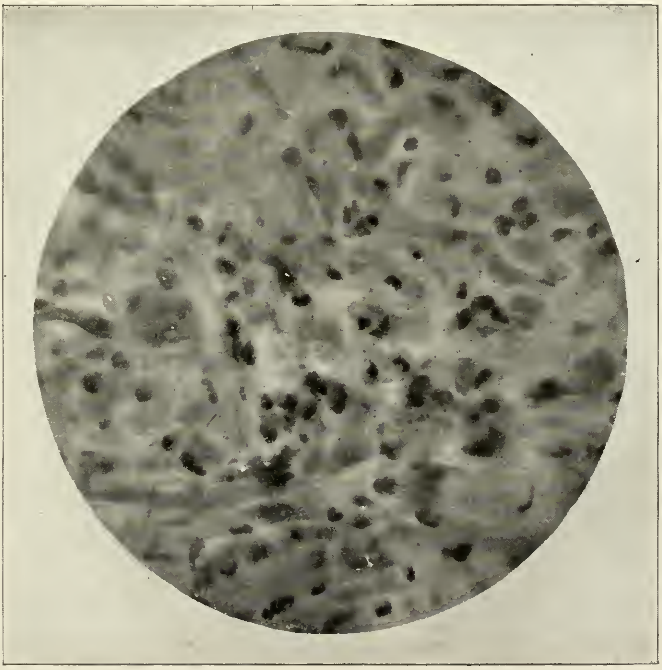
Proj. ¼ inch, ocular 1½ inch. Spencer.

FIG. 50.—LONGITUDINAL SECTION OF GINGIVAL BORDER, SHOWING ROUND-CELL INFLAMMATION DUE TO MERCURIAL POISONING.

the papillary layer of the sub-epithelial, mucous membrane, I instituted a series of experiments in mercurialization of dogs.

Dogs for the purpose were picked up in the streets. Some of these were operated upon by myself, but most of them were under treatment at the Post-Graduate Medical School. Care

was taken to secure those in health and with healthy gums. Mercury was introduced by the mouth, skin and hypodermic injection. It was no easy matter to get them under influence of the drug, since the power of the glands to eliminate the poison was enormous. In no case was salivation produced. The first symptom noticed was exhilaration, which would last from three days to a week. Then paralysis agitans would continue until death. In about a week the appetite would commence to fail and it was difficult to get the dogs to take food of any kind. The kidneys and bowels eliminated the poison. There was a



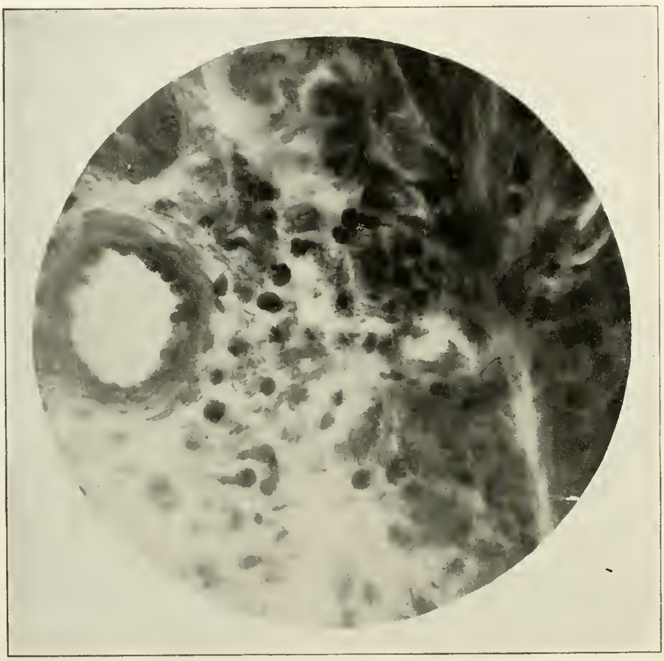
Pantachr. oil imm. 1/12 inch ocular. No. 3. Leitz.

FIG. 51.—LONGITUDINAL SECTION OF GINGIVAL BORDER. HIGHER MAGNIFICATION, SHOWING CONNECTIVE TISSUE INFILTRATION WITH PLASMA CELLS AND POLYNUCLEAR LEUCOCYTES. DOG.

rise in temperature. Some of the dogs died before gingivitis was observed. This demonstrated that not only does the nervous system become involved, but the organs of the body may be morbidly affected and death ensue before the gums show symptoms of disease. Some dogs were killed after the gums became

diseased. The time required to obtain results was from three to eight weeks. The age and physical condition of the dog caused this variation in time. After death the gum tissue was dissected from different parts of the jaws and placed in either fifty per cent alcohol, Muller's fluid, or two per cent formalin.

Sections of tissue from the gum margin and sides were made on a number of places. Some were imbedded in paraffin, others in celluloidin. The sections were stained according to various



Pantachr. oil imm. 1/12 inch ocular. No. 3. Leitz.

Fig. 52.—Longitudinal Section of Gingival Border. Higher Magnification, Showing Round-Cell Inflammation Extending to the Inner Coat of the Blood Vessel and also Plasma-mast Cells.

methods: Delafield's hæmatoxylin, eosin (Unna's), alkalin methylblue, carmin, Gramm's stain, etc.

Microscopic examination showed that the epithelial lining of the gums did not present pathologic changes, but appeared normal in every respect. Connective tissue below the gum epithelium (the tissue analogous to the papillary layer of the derma and the derma proper) presented unmistakable evidences of a

mild inflammatory process. There occurred in this connective tissue round-cell infiltration, generally moderate but in some places quite dense. This cellular infiltration extended from below (where it was densest) upward into the papillary layer (Figs. 50 and 51). The densest cellular infiltration usually occurred around the vessels (Fig. 51).

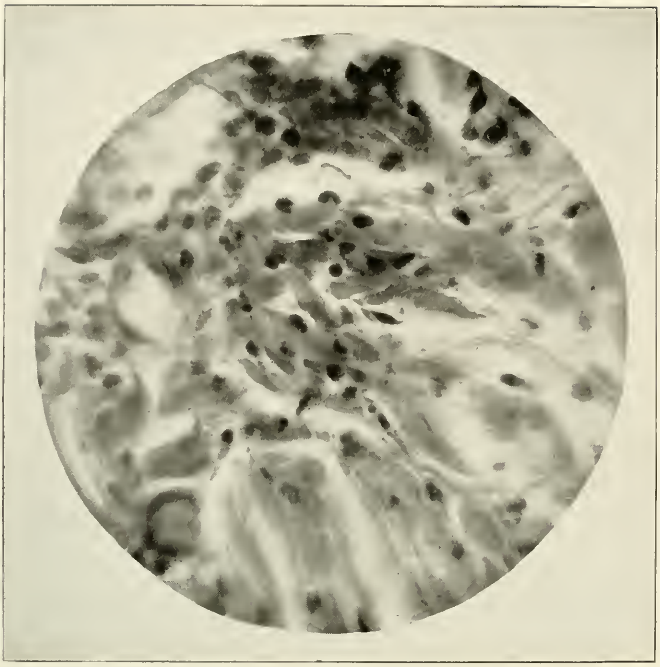
Under high magnification, the cellular infiltration was found to consist of polymorphonuclear leucocytes, plasma cells and

Projection 1/4 inch, ocular 1½ inch. Spencer.

FIG. 53.—LONGITUDINAL SECTION OF GINGIVAL BORDER, SHOWING ROUND-CELL INFILTRATION IN THE CONNECTIVE TISSUE AND EXTENDING INTO THE PAPILLAE. DOG.

plasma-mast cells, the latter with coarse basophilic granulations (Figs. 52 and 53).

In some places were seen between the round cells, short, broad fusiform cells, the protoplasm of which took quite well basic methylblue. These cells resemble very much fibroblasts and appear to be derivations of the plasma cells (Fig. 54). No bacteria were found either in the areas of cellular infiltration (inflammatory areas) or elsewhere. In these cases it is obvious



Pantachr. oil imm. 1/12 inch ocular. No. 3. Leitz.

FIG. 54.—LONGITUDINAL SECTION OF GINGIVAL BORDER, SHOWING ROUND-CELL INFLAMMATION DUE TO MERCURIAL POISONING. HIGHER MAGNIFICATION.

FIG. 55.

A monkey skull showing absorption of the alveolar process (original). The right central and left lateral have dropped out. The alveolar process is absorbed so that all teeth are loose.

that there had occurred a mild inflammation of the gums (gingivitis). While this could not be seen with the naked eye, microscopic examination demonstrated histologic features of an inflammatory process. The absence of bacteria justified the belief that this inflammation was not of microbic origin, but due to mercury, which by its well-known chemotactic influence produced the histologic changes of an inflammation.

Fig. 55 is the skull of a monkey who died aged one year of tuberculosis. Absorption of the alveolar process is the result of autointoxication acting upon a depleted organism. The right superior and inferior central and lateral incisors have loosened and dropped out. The roots of all the teeth are exposed to a marked extent. The teeth could be removed with the fingers.

CHAPTER XV.

RESEARCHES ON HUMAN IN INTERSTITIAL GINGIVITIS.

While hundreds of slides could be adduced in support of this chain of evidence, sufficient have been given to permit of the introduction of evidence from other phases of the subject.

The following autopsy was made by L. Hektoen on an old man, in whose case the pathologic diagnosis was as follows: Senile marasmus (senile emphysema, senile sclerosis of the aorta, atrophy of the parenchymatous organs), scurvy (hæmorrhagic gingivitis); chronic aortic and mitral endocarditis; fibrous myocarditis; chronic nephritis; caseo-calcareous areas in the right apex, spleen and left adrenal; double hydrothorax; bronchitis; fibroma of the stomach; amputation of the left lower extremity at the lower third of the thigh. The findings unrelated to the scope of the present investigation are omitted. The gums were found swollen, and here and there infiltrated with blood. There was purulent matter about the roots of the teeth, many of which were loosened and some of which could be removed with the fingers. The roots of the loosened teeth were covered with a granular grayish material.

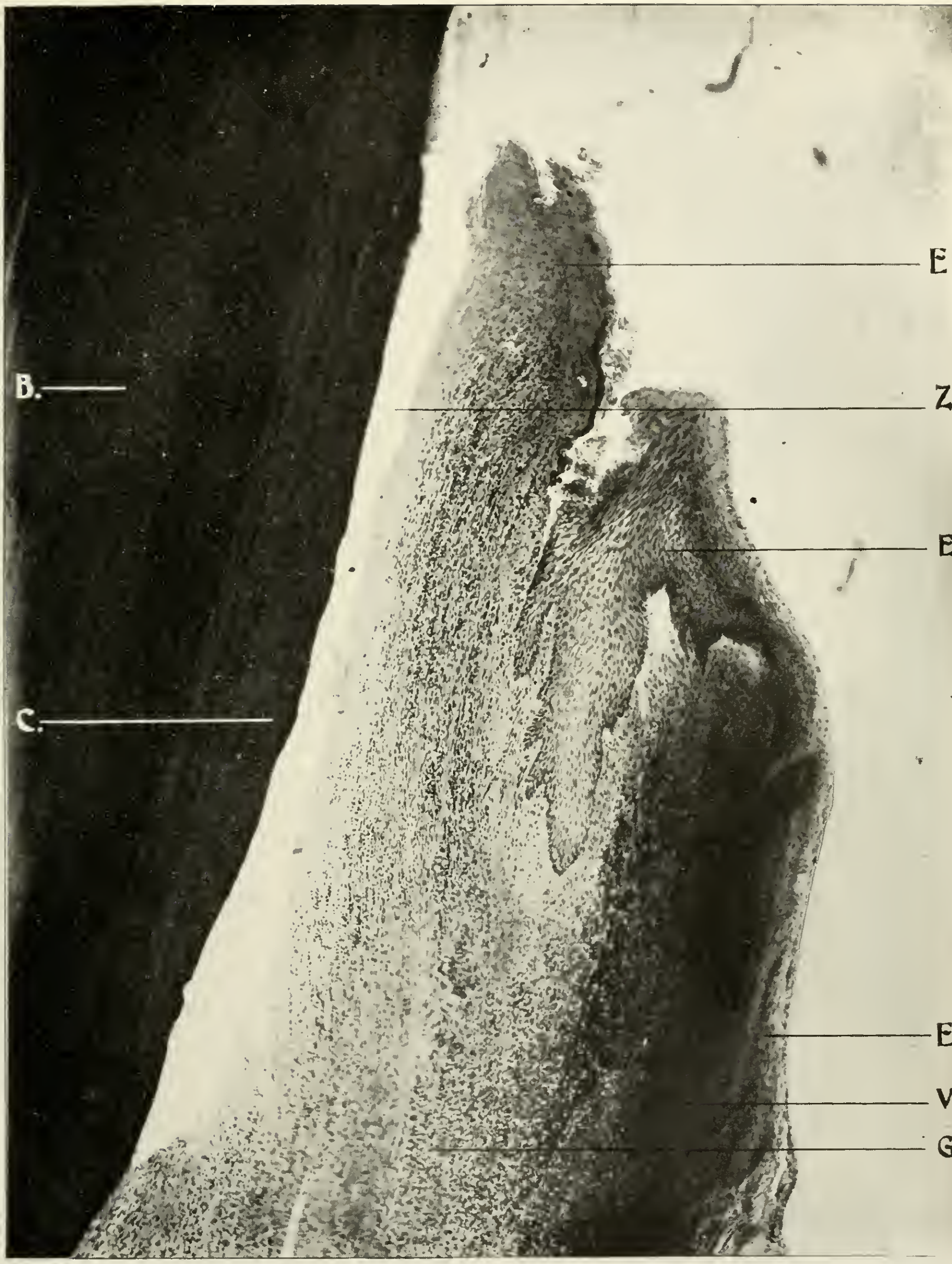
Bacteriologic examination of the root of the tooth gave the following results: Tube of bouillon from which agar plates were made, inoculated twenty-four hours before date, July 29, 1898. There were two varieties of colonies: Both grayish white. One kind is round, pin-head size, slightly elevated, with thin, wavy, but sharply defined border. Finely granular. Media inoculated from one of these. *Agar Slant:* White, tallow-like growth along the track of the needle, with thin, more translucent layer covering the rest of the surface. Only moderately elevated. Greenish tinge given to media. *Potato:* Elevated, "clumpy" growth, white on top, confined to needle track. Potato much darkened. *Blood Serum:* Gray, waxy growth, little elevated, sharply defined and thick border. *Gelatin Slab:* Saucer-shaped liquefaction at upper part, more tubular in deeper

portions. Flocculent masses throughout. *Glucose Agar:* Gas produced, white, thick growth on top. *Milk:* Alkaline, soft coagulation. *Bouillon:* Cloudy. *Characteristics:* Rapid growth, a sour, nauseating odor given off from all media. *Morphology:* Large coccus, single, in pairs and in groups. Stains easily by ordinary methods, also by Grams. The smaller colonies on agar plates (pin-point sized in center) with nearly transparent, illy defined peripheral zone. Border indistinct. Central portion in gray. Finely granular throughout. *Agar Slant:* Gray film over entire surface, somewhat thicker along the inoculation streak. At bottom there is a nearly white growth. Very light, greenish tinge to media. *Blood Serum:* Like on agar. *Potato:* Heavy dirty gray growth, wavy and sharply defined border. Looks like bunch of cauliflower. *Gelatin Slab:* Liquefied, saucer-shaped at top, tubular in deeper part. Growth mostly in upper stratum. *Lit. Milk:* Negative. *Bouillon:* Cloudy. *Glucose Agar:* Gas produced. *Characteristics:* Rapid growth, stinking odor from all media. *Morphology:* Small, slender bacilli; actively mobile, spores. Takes ordinary stains readily and is not decolorized by Gram's method.

Only the lower frontal teeth and corresponding part of the jaw could be examined. The epithelial covering of the gums appeared to be quite intact. In some places it was a little thickened, and its lower layers infiltrated with new cells. The subepithelial tissue was much thickened, presenting the general structure of an inflammatory granulation tissue of some standing. Areas occurred in which there were many new cells and but little stroma. In other foci the tissue was more fibrous, the new cells running in bands. Here and there occurred free and intracellular granular, yellow pigment. Typical hyaline bodies of various sizes, and staining a precise bluish violet with Gram's method, were found in rather small numbers. In some places small sub-epithelial abscesses were met with, which (in the instance of a district including a lower incisor) were really subperiosteal. The contents consisted of nuclear detritus and bacteria (mostly cocci) which have accumulated, especially upon and in the walls of the minute cavities extending from such an abscess. There seems to be a complete occlusion of the vessels

X 40. 35 M. M. Zeiss. Micro-photograph, reduced one-third.
FIG. 56.—LONGITUDINAL SECTION OF TOOTH, ALVEOLAR PROCESS AND GINGIVAL BORDER, SHOWING ACTIVE INFLAMMATION IN SCURVY IN MAN.

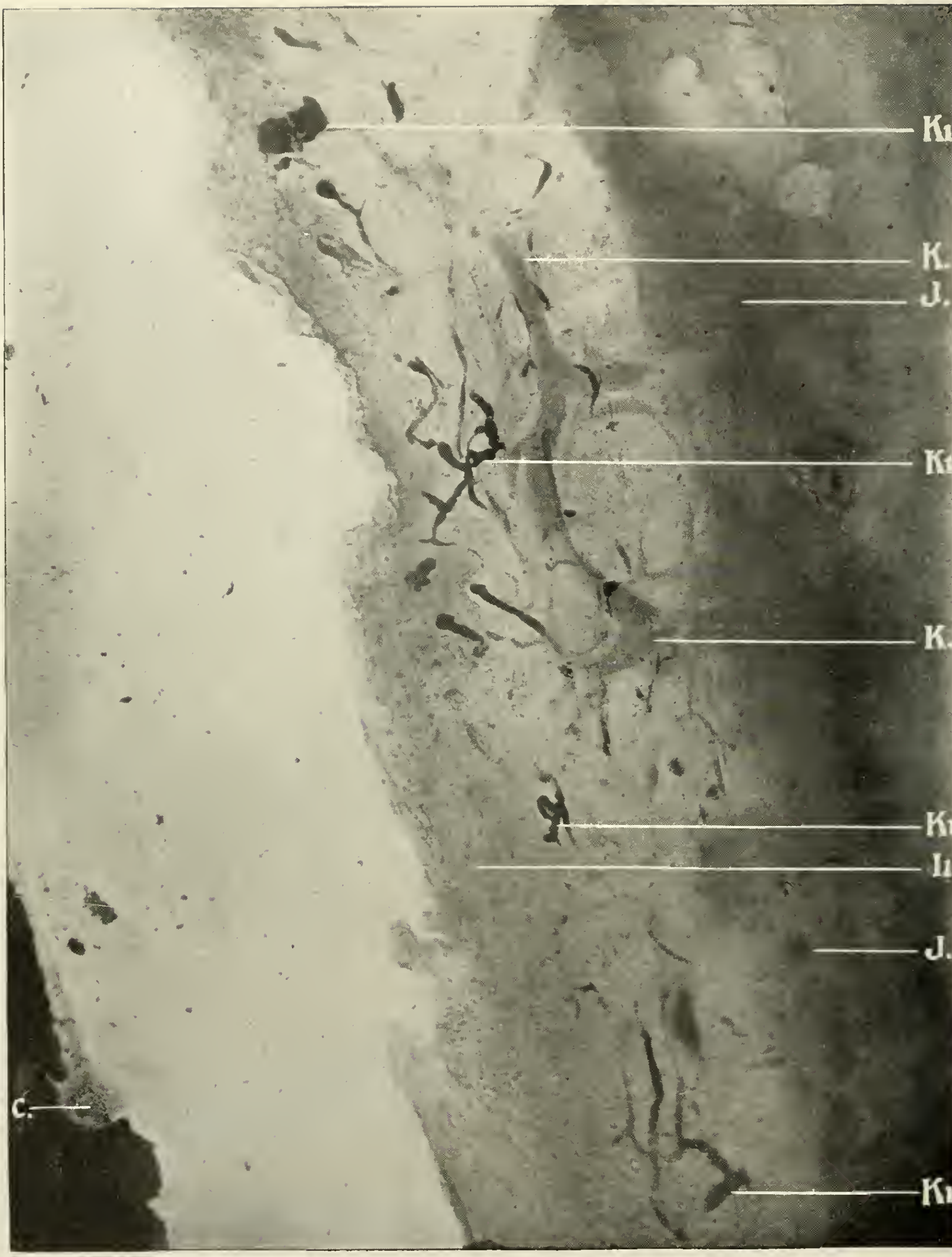
B, Dentine. C, Cementum. E, Epithelial Tissue. G, Submucous Membrane. H, Periosteum. J, Alveolar Process. L, Haversian Canals. M, Fibrous Tissue. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. RR, Space Pocket from Want of Union of the Epithelial Fold.



X 40. 35 M. M. Zeiss. Micro-photograph, reduced one-third.

FIG. 57.—LONGITUDINAL SECTION OF A TOOTH, ALVEOLAR PROCESS AND GINGIVAL BORDER, SHOWING ACTIVE INFLAMMATION IN SCURVY IN MAN.

B, Dentine. C, Cementum. E, Epithelial Tissue. G, Submucous Membrane. V, Violent Inflammation. Z, Sloughing of the Epithelial Tissue Due to Calcic Deposits. AA, Point of Union of Epithelial Tissue and Peridental Membrane.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 58.—Longitudinal Section of Tooth, Alveolar Process and Peridental Membrane, Showing Blood Pigment in Blood Vessels of Peridental Membrane in Scurvy in Man.

C, Cementum. J, Alevolar Process. K, Capillaries. I^1, Inflamed Peridental Membrane. K^1, Blood Pigment in Capillaries.

X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 59.—Longitudinal Section of Tooth and Gingival Border, Showing Active Inflammation Extending through the Mucous and Peridental Membranes. Scurvy in Man.

B, Dentine. C, Cementum. E, Epithelial Tissue. V, Violent Inflammation. AA, Point of Union of Epithelial Tissue and Peridental Membrane. RR, Space Pocket from Want of Union of Epithelial Fold. M^1, Inflamed Fibrous Tissue.

(capillaries) with typical bacteria masses, staining a peculiar bluish violet color with hematoxylin, and blue with Gram's method, so that the vessels presented the appearance of being very successfully filled by an infection mass: the small dilatations, the branches and the larger vessels (judging from structure these seemed to be veins) were sometimes brought out very nicely. The intravascular growth of bacteria extended into the bone below as well as, and more especially into, the peridental membrane.[1] These abscesses (suppurative periostitis) occur almost exclusively upon the inner surface of the alveolar process, being confined (as far as there was occasion to observe) to the external aspect of the process. There was always a thin, sound layer of bone separating the abscess from the peridental membrane. Very generally the spaces in the adjacent bone were filled with a cellular fibrous tissue in which occurred islands of osteoid tissue. The bone trabeculæ were generally covered by a thin layer of osteoid tissue, which (from the greater number of cells it contains, as compared with the other bones) must be newly formed. Rows of osteoblasts were found often upon the trabeculæ. Few Howship's lacunæ were found, and these were filled with small cells. There were no osteoclasts in the areas about the abscesses. The bone outside of the alveolar process was quite unchanged.

The "bacterial thrombosis" not unusually extended into the peridental membrane, which then refused to stain as clearly as normal. The upper part of the peridental membrane was usually the seat of cell proliferation, and of the formation of fibrous tissue, due to the direct extension of the similar process in the sub-epithelial connective tissue of the gingivus. There were no indications that the process began below, at the apex of the tooth, for example, and extended upward. In the peridental membrane, and often connected with the cementum of every tooth examined, were very many so-called calcospherites; calcified, concentrically lamellated, round or oval bodies, not unlike the "corpora amylacea." In many instances, it seemed as if the

---

[1] The abscesses have a definite outline or wall of ordinary cellular fibrous tissue displaying striking evidences of active inflammation. The tissue about the capillaries filled with bacteria refuse to stain clearly, but there are no signs of inflammation.

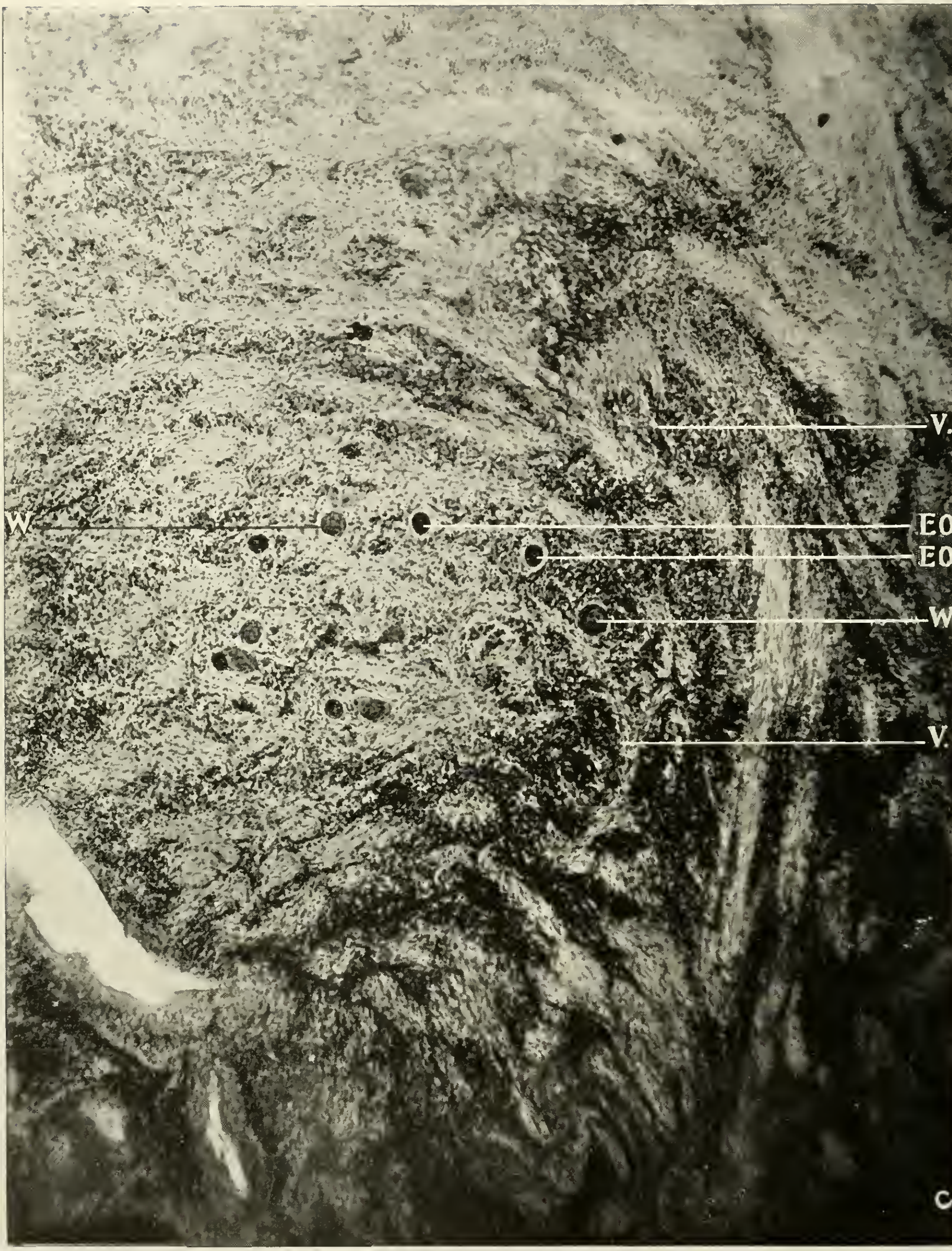
body had formed in the cement or at its margin—the cement presenting here a nodular condition.

Fig. 56 illustrates a section through the tissues of the jaw and cuspid tooth. The epithelium is not so dense and thick as in a similar section from the dog. Inflammation extends along the papillary layer of the submucous membrane (G) and involves the deeper structures. The mucous membrane layer has doubled upon itself, forming a pocket (R R). Violent inflammation is evident at V. This is of unusual interest, since it demonstrates that inflammatory products may be carried by the blood vessels anywhere throughout the alveolar process, and may result in abscesses. The inflammation extends throughout the periosteum (H), the fibers of which extend from the root of the tooth over the border of the alveolar process (J). There the coarse fibers of the periosteum contrast decidedly with the finer fibers of the sub-epithelium. Absorption and contraction of the alveolar process (fully one-half the length of the root of the tooth) has taken place, as well as lateral absorption. The inflammatory process extends through the Haversian canals (L).

Fig. 57 is a section through the jaw at the lateral incisor. The epithelium (E) is seen upon the outer surface of the alveolar process as far as the root of the tooth. The inner fold next to the tooth has disappeared through encroachment of deposits which have been destroyed by nitric acid. Inflammation extends throughout the entire submucous membrance (G). The most marked inflammation in this case is entirely upon the outer border (V).

Fig. 58 shows a section of the peridental membrane (I) and alveolar process (J). Capillaries (K) interlace through the field, the largest number being distributed along the alveolar wall. Blood pigment containing bacteria is noticeable (K′).

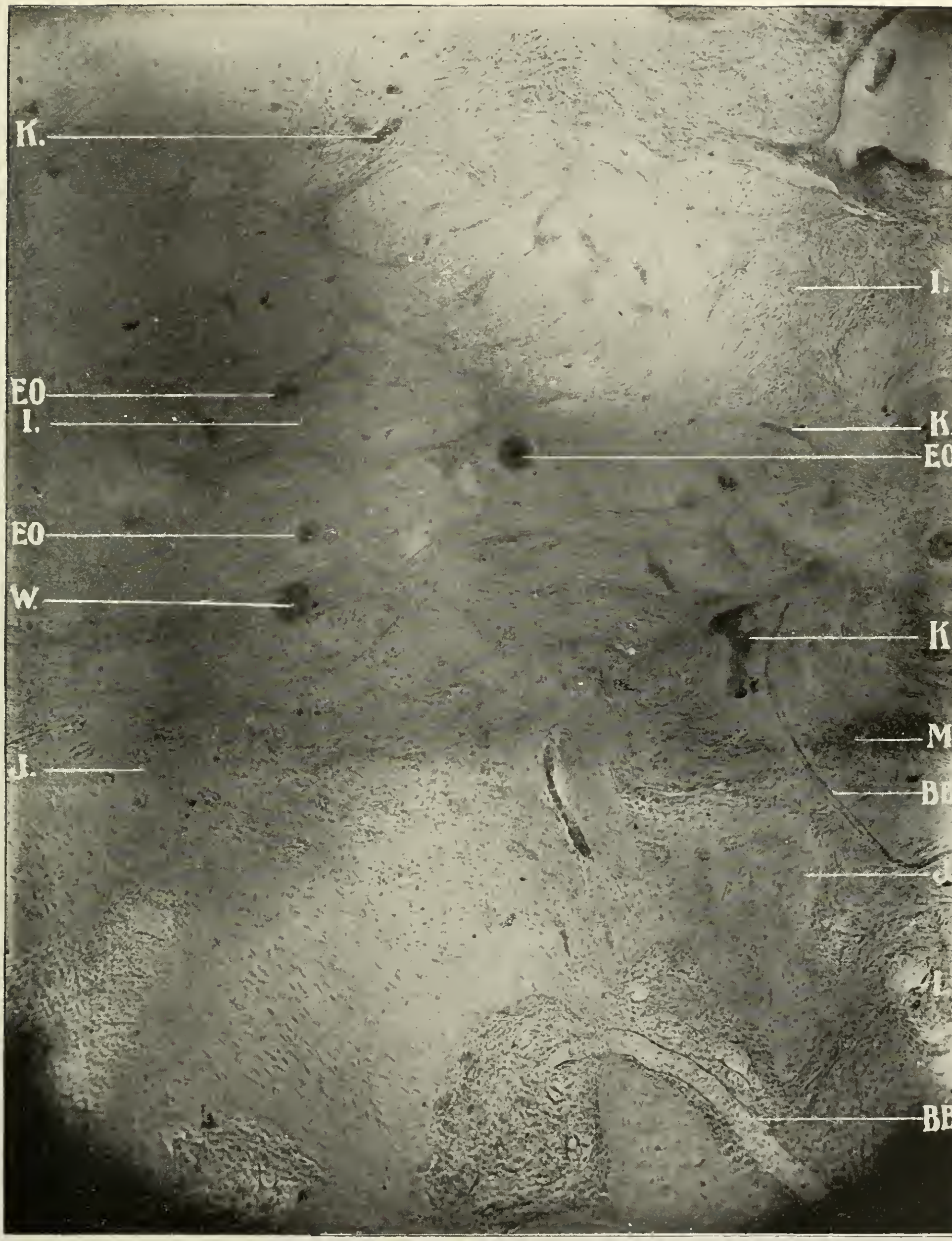
Fig. 59 is an amplification of a section depicted in Fig. 56. This gives a clearer idea of the folding of the epithelium (E) and submucous membrane (G) layer upon itself, forming a pocket (RR), in which may be seen an accumulation of food and bacteria. It also shows extensive inflammation throughout the entire field. Marked inflammation is evident at V. The point



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 60.—Cross Section Peridental Membrane, Showing Active Round-Cell Inflammation. Scurvy in Man.

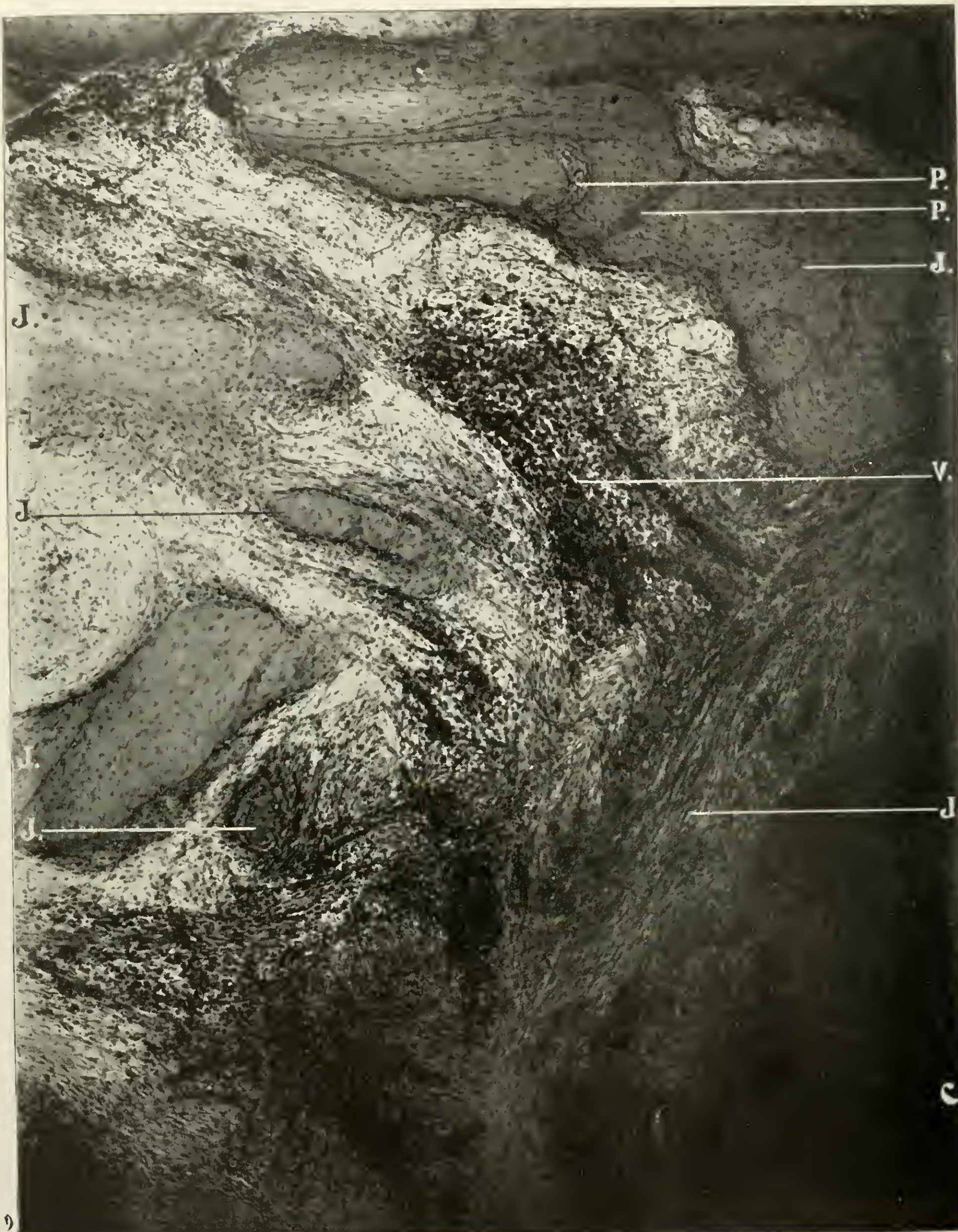
C, Cementum. V, Violent Inflammation. W, Epithelial Débris. EO, Endarteritis Obliterans.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

Fig. 61.—Cross Section of Inflamed Peridental Membrane. Scurvy in Man.

I, Peridental Membrane. J, Alveolar Process. K, Capillaries. L, Haversian Canals. BB, Blood Vessels of Von Ebner Preceding Perforating Canals. EO, Endarteritis Obliterans. W,-Epithelial Débris.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

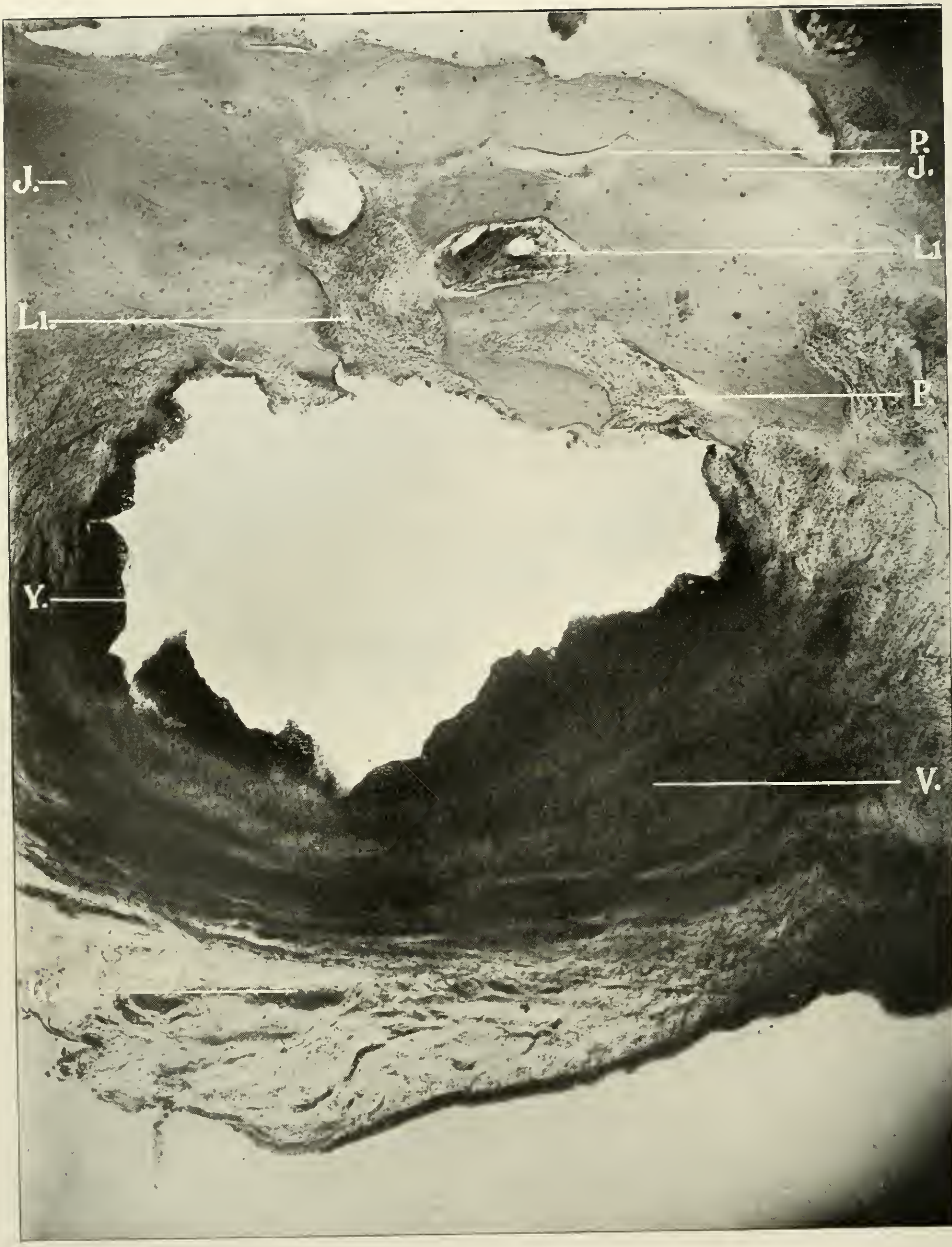
Fig. 62.—Cross Section of Tooth, Alevolar Process and Peridental Membrane, Showing Active Inflammation and Absorption of Bone. Scurvy in Man.

C, Cementum. I, Peridental Membrane. J, Alveolar Process. P, Perforating Canal Absorption. V, Violent Inflammation.

X 40. 35 M. M. obj. Zeiss. Micro-photograph, reduced one-third.

FIG. 63.—CROSS SECTION OF PERIDENTAL MEMBRANE AND ALVEOLAR PROCESS, SHOWING ACTIVE INFLAMMATION AND ABCESS. SCURVY IN MAN.

J, Alveolar Process. T, Bacteria. Y, Abcess. I[1], Inflamed Peridental Membrane. L, Inflammation Extending through Enlarged Haversian Canals.



X 75. A. A. obj. Zeiss. Micro-photograph, reduced one-third.

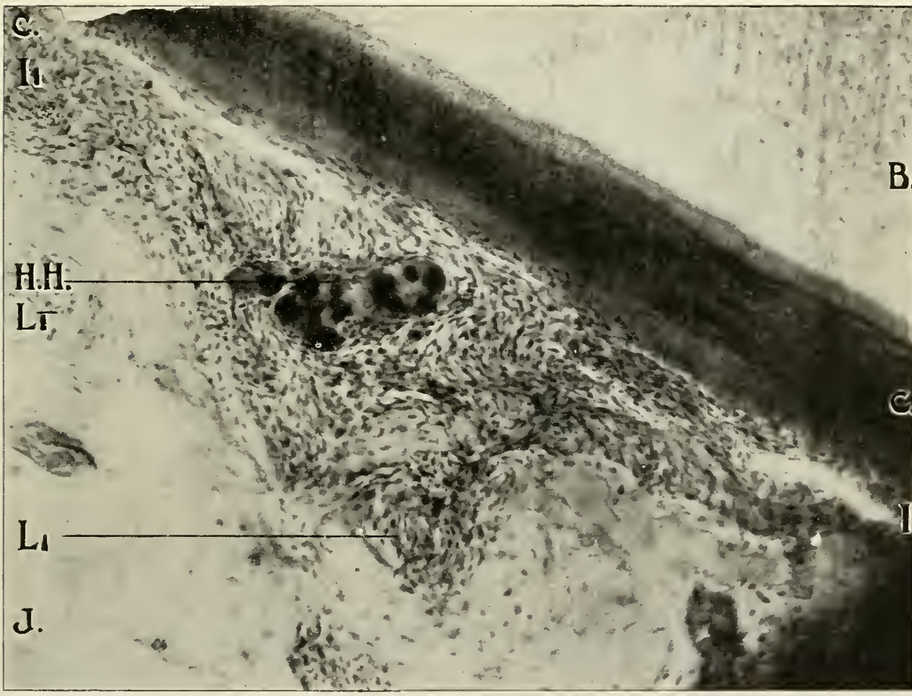
Fig. 64.—Cross Section of Peridental Membrane and Alveolar Process, Showing Active Inflammation and Another Larger Abcess. Scurvy in Man.

J, Alveolar Process. P, Perforating Canal Absorption. V, Violent Inflammation. Y, Abcess. I[1], Inflamed Peridental Membrane. L[1], Inflammation Extending through Enlarged Haversian Canals.

of union of the sub-epithelial layer and the periosteum is shown (AA).

Fig. 60 illustrates inflammation of the peridental membrane with epithelial debris (W) scattered over the field. Endarteritis obliterans (EO) is also noticed at various positions. Marked inflammation may be seen at V.

Fig. 61 illustrates a section of the peridental membrane (I) and alveolar process (J) with inflammation extending throughout. Capillaries (K) are also noticeable in large quantities,



X 300. No. 2 projection ocular. D. D. obj. Zeiss.

Fig. 65.—Cross Section of Tooth, Alveolar Process and Peridental Membrane, Showing Active Inflammation with Calcospherite in Membrane. Scurvy in Man.

B, Dentine. C, Cementum. I, Peridental Membrane. J, Alveolar Process. HH, Calcospherite. J^1, Inflamed Peridental Membrane. L^1, Inflammation Extending through Enlarged Haversian Canals.

nearer the alveolar process than the root of the tooth. Epithelial debris is evident at W. Endarteritis obliterans (EO) may be seen in different portions of the field. Inflammation has extended into the Haversian canals (L) but absorption has not

occurred to any great extent. The blood vessels of Von Ebner (BB) are quite well shown.

Fig. 62 is a section showing the cementum (C), the peridental membrane (I) and the alveolar process (J). Marked inflammation extends through the peridental membrane, thence through the Haversian canals (which are entirely obliterated). Absorption of the trabeculæ (halisteresis) has resulted to the extent that what remains of the alveolar process (J) are islands of bone held in place by the fibrous tissue. Blood vessels of Von Ebner with perforating canals are seen at P.

Fig. 63 shows a section of the peridental membrane and alveolar process with a large abscess originally within the alveolar wall. Inflammation spreading through the peridental membrane has occurred at I′, while the decalcified alveolar process is also shown (J). Violent inflammation has taken place within the alveolar wall, and an abscess (Y) has formed. The wall of the abscess is distinctly seen, with masses of bacteria (T) clinging to the inner sides. The process of halisteresis (Q) (bone decalcification) is seen as a result of the violent inflammation. The entire wall next to the peridental membrane and about the abscess has been destroyed, and the different stages in the process by which this has been accomplished are beautifully shown.

Fig. 64 illustrates a larger abscess (Y) from another location. This is also situated within the alveolar wall, showing that the inflammatory products extend through the blood vessels. Marked inflammation is seen upon the side next to the peridental membrane (I′), while rapid absorption—halisteresis (Q) and perforating canal (P)—is proceeding at the borders of the abscess and nearest the alveolar process.

Fig. 65 shows a section of a tooth (B and C), inflamed peridental membrane (I′), with absorption of the alveolar process (J). In the inflamed peridental membrane may be seen a calcospherite, oblong in form.

INTERSTITIAL GINGIVITIS IN MAN FROM DRUG ACTION.

A forty-eight-year-old merchant was dyspeptic, debilitated and asthmatic, and for the treatment of these conditions he had been under calomel and tonics for a little less than two weeks.

When he came under observation, the mucous membrane and gums were then much inflamed. There was marked sialorrhœa. The teeth were loose. The gums were swollen. Pus oozed from the gums. The breath had a decided metallic odor. At my suggestion, his medical attendant stopped the calomel. He was then ordered six pints of spring water daily. The gums were, on alternate days, saturated with iodin. In a few days the soreness and swelling were so reduced that the deposits could be removed. The patient was discharged cured in a short time other than as to the right inferior second molar, which was so loose as to require removal. This tooth was placed immediately

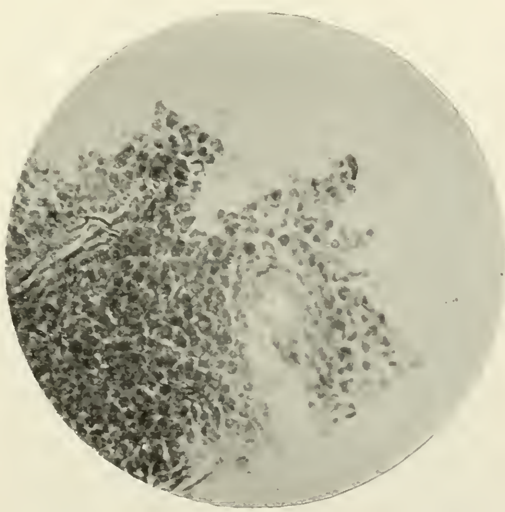


FIG. 66.—SHOWS A SMALL FRAGMENT OF INFLAMED PERIDENTAL MEMBRANE.

in fifty per cent alcohol for twenty-four hours and then removed to absolute alcohol for twenty-four hours more. The membranes had receded about two-thirds the length of the root. Sections for microscopic purposes were made from the lower third of the root. Of these sections, Fig. 66 shows a small fragment of inflamed peridental membrane. Fig. 67 exhibits violent round-cell inflammation, degeneration and liquefaction of tissue.

A thirty-five-year-old diabetic painter came under observation for plumbic poisoning. His gums were swollen. There was decided sialorrhœa. The teeth were loose. Pus flowed from the gums. He was placed on ozonate spring water and the gums

were saturated with iodin on alternate days. Three loose teeth were removed and placed in alcohol. Sections from the upper third of the left superior second bicuspid gave results on microscopic examination similar to those already described as occurring in mercurial poisoning. Fig. 68 shows round cells of inflammation. Fig. 69 illustrates very marked degeneration of the peridental membrane. In the lower right-hand corner are seen the root of the tooth, dentine and cementum. The whole surface of the peridental membrane is in an advanced phase of

Fig. 67.—Violent Round Cell Inflammation, Degeneration and Liquification of Tissue.

inflammation. Just at the border of the root is evident an area of membrane softening. Just beyond, but joining, is noticeable breaking down of tissue. In the center are seen two areas of softened tissue more advanced in degeneration.

One occupation disease which has been ignored in the etiology of interstitial gingivitis is "brass-workers' ague." In almost all brass-workers, a stain varying from a bright to a brownish green is detectable on the necks of the teeth between the crowns and

the gum insertion. This is most obvious in the upper jaw. After a while, as E. Hogben[2] has shown, the teeth become loosened and fall out. Before these changes in the gum occur nervous symptoms have developed from the brass poisoning.

Arsenic should be taken into account in the etiology of interstitial gingivitis. This drug has a very decided tendency in certain subjects to cause, even in small doses, marked stomatitis and irritation of the mucous membranes throughout the body.

Tartar emetic and the other preparations of antimony, producing irritation of the mucous membranes of the mouth and elsewhere, may act as predisposing and exciting factors of interstitial gingivitis.[3]

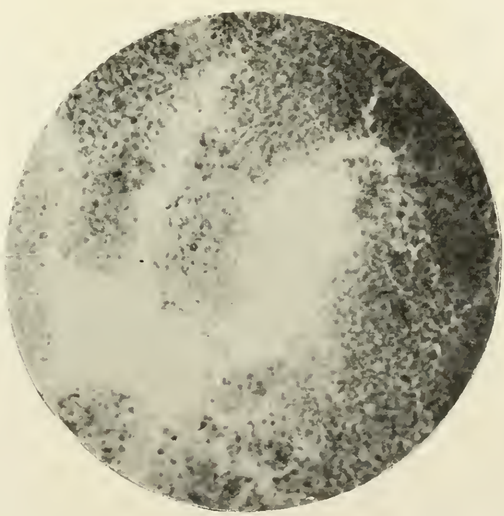


FIG. 68.—SECTION OF PERICEMENTAL MEMBRANE SHOWING ROUND CELL INFLAMMATION.

Among the drugs which should be taken into account in the etiology of interstitial gingivitis is potassium bromide. This produces in certain individuals, or when given to excess, marked increase of the saliva with irritation of the mucous membranes of the mouth, followed later by dryness of the mouth and shrinking of the gums. The bromides have, as H. C. B. Alexander[4] has shown, a tendency to irritate all the mucous membranes of the body as well as the skin. Therefore, in dealing with cases

[2] Birmingham Medical Review, 1887.
[3] Lewin: Untoward Effects of Drugs.
[4] Alienist and Neurologist, July, 1896.

of interstitial gingivitis in which the bromides are being taken, this factor should not be neglected. In these cases the symptoms due to the bromides are apt to be charged to the nervous state for which the bromides have been given. The irritation of the mucous membrane by the bromides may occur quite early among

FIG. 69.—MARKED INFLAMMATION AND DEGENERATION OF THE PERIDENTAL MEMBRANE SHOWING FOUR PERIDENTAL ABSCESSES.

the untoward effects produced by them. In all probability the bromide rather than the alkali is the source of these untoward effects.

What is true of the bromides is also true to an even greater degree, as has elsewhere been shown, of the iodides.

CHAPTER XVI.

RESEARCHES ON HUMAN IN PERICEMENTITIS.

Referring to Chapter VI we find that the peridental membrane is a fibrous tissue situated between the root of the tooth on the one hand and the alveolar process on the other. It covers the root of the tooth. This structure, to all intents and purposes, is a continuation of the trabeculæ or fibrous tissue in the alveolar process. In other words, the alveolar process is nothing more or less than fibrous tissue filled in with lime salts.

If a piece of jaw containing teeth be placed in a weak acid solution and the lime salts dissolved, there would be no distinction, under the microscope, in the remaining structure from the root of the tooth to the outer surface including the periosteum except in quality of fibers. When a low form of inflammation is set up in the alveolar process, the lime salts are absorbed, leaving the fibrous tissue in like manner. If on the other hand, the inflammation is more intense, the trabeculæ or fibrous tissue is also destroyed.

Irritations of a local nature first produce a gingivitis of the gum tissue which extends along the blood vessels into the deeper tissues and assumes an interstitial character. When the irritations are constitutional, due to autointoxication, drug, or other irritation and poisons, the deeper tissues become involved, then assuming an interstitial character, later affect the gums, producing gingivitis. When the peridental membrane becomes involved, whether from local or constitutional causes, it is termed pericementitis. This disease is always recognized by soreness and a slight elongation of the tooth or teeth.

If the cause be slight or if removed, the membrane will return to its normal condition. On the other hand, if the cause be severe and of long standing the inflammation will become chronic, resulting in absorption of the alveolar process and perhaps abscess.

A low form of pericementitis may act upon the surrounding tissues in different ways, depending, to a great extent, upon the nervous system. First, if there are no corresponding teeth for antagonism on the opposite jaw, the inflammation will cause a deposition of lime salts in the alveolar process, causing elongation which continues until the tooth or teeth meet resistance. Second, exostosis of the cementum of the tooth. Third, the inflammation results in peridental or alveolar abscess. Later, especially if the irritation be of a constitutional nature, the inflammation will cause absorption of the alveolar process and exfoliation of the teeth.

A seamstress bites her thread, pericementitis results. If treatment be resorted to and the habit stopped, the peridental membrane will be restored to health. If, however, this habit be continued, interstitial gingivitis results, with absorption of the bone and loosening of the teeth. The habit of biting threads with the teeth causes an extra amount of work upon the peridental membrane. Persons suffering with autointoxication would naturally find such teeth first involved in interstitial gingivitis. The same results follow when a low form of inflammation occurs in the three classifications just mentioned above. Persons of low vitality, poorly nourished people suffering with prolonged sickness and pregnant women have periostitis and general interstitial gingivitis. Persons overworked or suffering with neurasthenia are prone to it.

In syphilis, pericementitis and interstitial gingivitis are set up and not only in the alveolar process, but all bones of the body may become involved, causing hypertrophy as well as absorption and death of bone. Heat and allied irritation will produce interstitial gingivitis and bone absorption.

Some more severe forms of pericementitis and interstitial gingivitis deserve attention from the irritation point of view. I have for years moved the teeth of dogs with regulating appliances, using a screw with sixty threads to the inch. In some the screw was turned one-fourth round, in others one-half, and in still others one full turn once a day. Some of the dogs were killed in three days, others in a week, and still others in two weeks. By this method the simplest and most severe forms of

pressure were applied, the length of time being brief as well as extended. These tissues were decalcified, cut, stained and mounted for the microscope. In every case inflammation of the pericementum was produced. This disproves the theory so long held, that bone absorption in regulating teeth is purely a physiologic process. Teeth were also extracted from dogs and after a week they were killed. The bone was decalcified, cut, stained and mounted for the microscope. The absorption was inflammatory in character. The jaws of dogs and monkeys who were erupting the permanent teeth were treated in like manner. Ab-

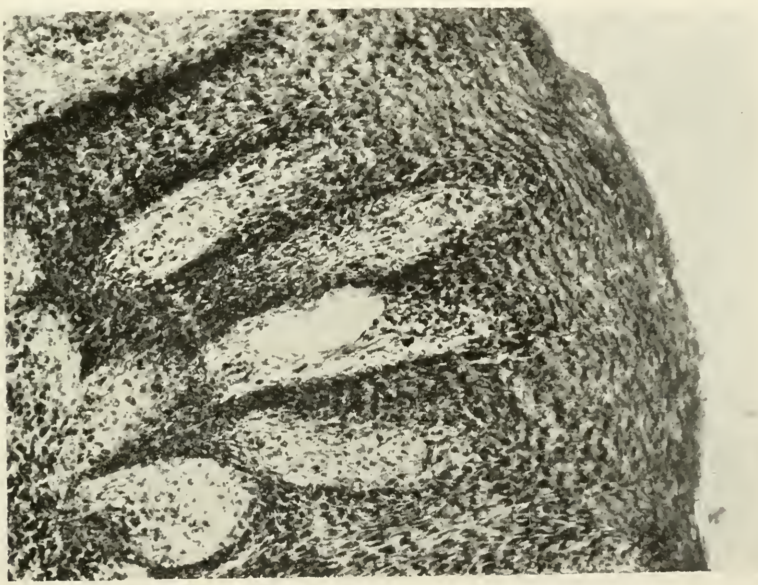


FIGURE 70.
Inflammation of the gum margin (original).

sorption of the alveolar process to allow the teeth to pass into position was of inflammatory type. Simple irritation, as well as severe pressure, hence produces the same pathologic process, pericemental inflammation and interstitial gingivitis.

The blood vessels which supply the gums, pericemental membrane and alveolar process are, as I have elsewhere demonstrated closely connected. Those in the pericemental membrane form a plexus along the wall of the alveolar process, while only a small number are near the roots of the teeth. So closely con-

nected are these that the vessels in one cannot become involved without affecting those of the other tissues. Hence, gingivitis and pericementitis occur which in reality become interstitial, or interstitial inflammation appears, which in reality becomes gingivitis. No matter what the cause may be, or whether the initial lesion be in the gum or interstitial structure, absorption of the alveolar process eventually results.

I began my experiments upon the peridental membrane in 1896. In 1897, I read an article before the Section on Stomatology of the American Medical Association, demonstrating the

FIGURE 71.

Section deeper at the alveolar border (original). Active inflammation around two arteries which are becoming thickened.

pathology of the peridental membrane from simple inflammation to the breaking down of tissue and the formation of abscess.

A bicuspid tooth with a gold crown attached was taken from the mouth of a fifty-four-year-old man. He was suffering from autointoxication and neurasthenia. The tooth had become quite loose although the gum tissue was still intact. The irritation was both local and constitutional in character, since the gold

crown irritated the gum margin and a slight attack of Bright's disease caused self poisoning.

The following illustrations are taken from microscopic slides and magnified four hundred and eighty diameters. Fig. 70 shows the gum tissue. The epithelial layer shows the dipping down of the legs into the true mucous membrane below the basement membrane, with round cell infiltration due to irritation. Fig. 71 is a cross-section of peridental membrane of the left inferior central incisor of a lady twenty-nine years of age who

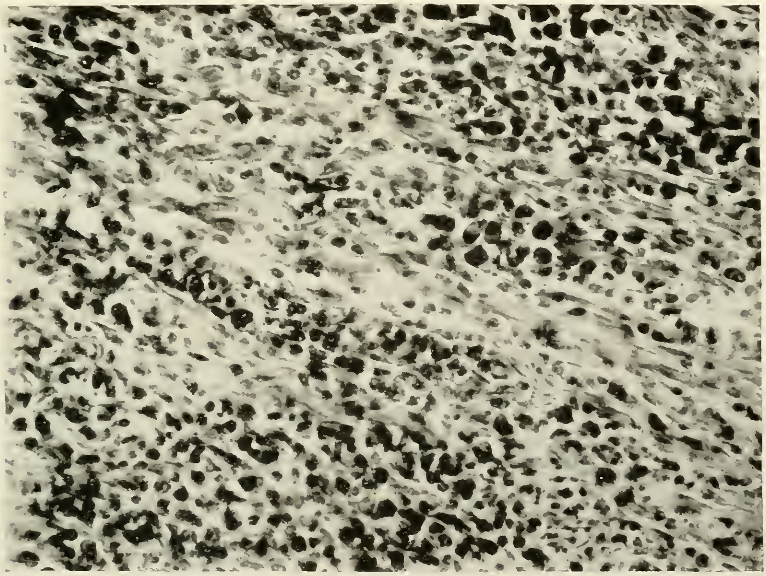


FIGURE 72.

Active inflammation in peridental membrane and trabeculæ (original).

had been under my care for fourteen years. She was in the habit of biting her thread with this tooth. Her occupation, that of dressmaking, gave her little or no exercise and she was also overworked. She drank no water; she was suffering from sleeplessness and nervousness due to indigestion and autointoxication. Sections of tooth, after decalcification, were made in the usual manner for the microscope. A cross-section shows two blood vessels which are considerably thickened (endarteritis obliterans) in its early stages with round cell infiltration in the tissues about them.

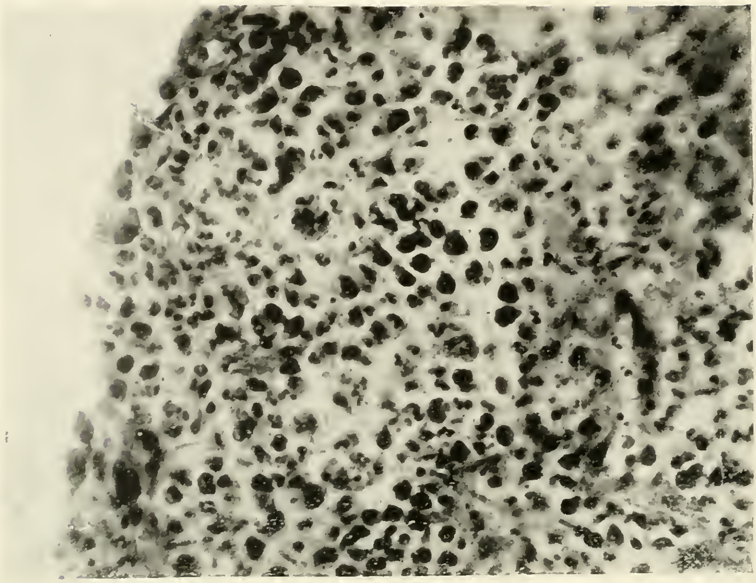


Figure 73.

Violent inflammation in the peridental membrane and trabeculæ (original).

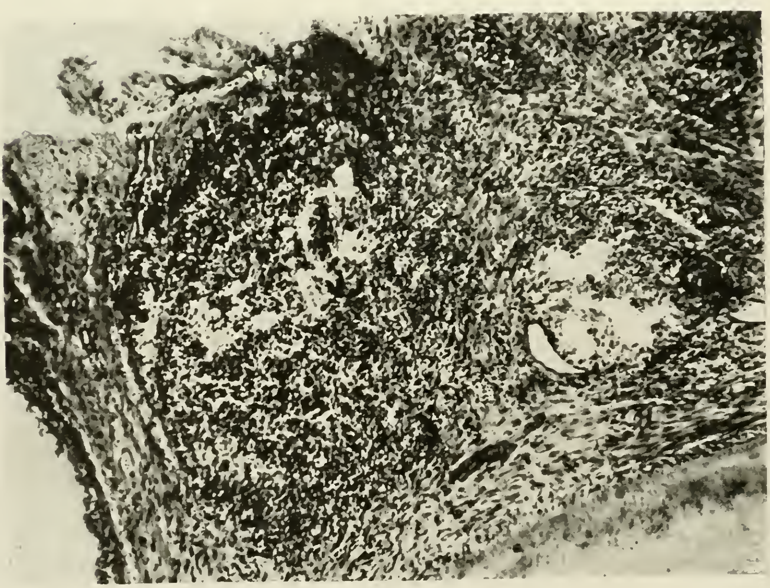


Figure 74.

Shows the root of the tooth, the peridental membrane, active inflammation in the trabeculæ and the formation of two abscesses. Note that both these abscesses are located in what was once the alveolar process. The peridental membrane can be readily observed between the root of the tooth and the nearest abscess (original).

The preceding three illustrations show the different stages of inflammation and liquefaction of the peridental membrane of the right superior first molar in a forty-year-old lady, a marked neurasthenic who has had periostitis and interstitial gingivitis with pyorrhœa alveolaris for the last twenty years and is now losing her teeth very rapidly. Fig. 72 shows a cross-section of palatal root near the apex, showing active inflammation in the peridental membrane. The round cell inflammation with exudate is rapidly collecting between the bundles of connective tissue fibers. Fig. 73 is a cross-section of the same root nearer the apex showing connective tissue with active inflammation, a stage further advanced than that of the previous illustration. In this area no fibrous tissue can be seen. Fig. 74 shows a still further advance in degeneration and liquefaction, forming an abscess. This is a lower magnification, showing a portion of the two buccal roots of the tooth, peridental membrane and the trabeculæ or fibrous tissue which was once alveolar process between them. The peridental membrane may easily be distinguished around each root from the trabeculæ between them. Two areas of softening and liquefaction may be seen forming two abscesses.

CHAPTER XVII.

LOCAL CAUSES OF INTERSTITIAL GINGIVITIS.

The local irritations producing interstitial gingivitis are the eruption of the teeth, change in function, tartar, uncleanness, lactic acid ferment, irritations due to modern dentistry, irregular teeth, regulating teeth, implantation of teeth.

Many years ago[1] I stated that modern dentistry was one of the most fruitful sources of interstitial gingivitis. Irritations from foreign substances, such as detached bristles of the toothbrush, too great friction in brushing the gums and alveolar process, where the latter is prominent, injudicious use of the toothpick, the use of ligatures in holding in rubber dam and regulating teeth, regulation of teeth with any applicance, application of the rubber dam, the use of clamps, crown and bridge work, irritation and heat due to artificial dentures and regulation plates, overlapping fillings, injuries from instruments, the devitalization of pulps, root fillings which throw increased work upon the peridental membrane or irritate it, in a word whatever irritates the gum margin, peridental membrane or alveolar process, is likely to produce inflammation which later becomes chronic. I am convinced that the disease is contagious, or progressive, not from one individual to another, but from one tooth to another in the same mouth.

The greatest and most important local cause of interstitial gingivitis is that of the eruption of the teeth since nearly every person possesses two sets.

Tooth eruption of both the first and second set ushers in the second and third periods of stress. It is at these periods when great changes take place in the system of the child and mark the early eras of future welfare. The absorption of bone in the eruption of the teeth and the building up of bone about the roots

---

[1] Talbot, The Dental Cosmos, Nov. 1886.

to hold them in place is an inflammatory process. How well this is accomplished depends upon the health of the child. In neurotic and degenerate children, when the nervous system is unstable and the child's vitality is of low order, the process of absorption and deposition of bone cells is not carried on normally. Especially is this true in the building up of the alveolar process about the roots of the teeth. Interstitial gingivitis remains in the alveolar process to a greater or lesser extent during the entire period of and until the shedding of the first set of teeth. The extreme illustrations of this disease are those connected with rachitis, infantile scurvy, inherited syphilis, marked neurosis, degeneracies and similar diseases. The jaws are often small, the alveolar processes are undeveloped and quite loosely built up. The gums are always inflamed and frequently ulcerated. At the second period of stress, when the first teeth are erupting the entire alimentary canal as well as all the internal organs undergoes changes in preparation for the reception of solid starchy food. The effects upon nutrition at this time are severe. In every patient there is more or less inflammation in the alveolar process, gums and peridental membrane until the first teeth are shed.

When the second set of teeth erupt there is a double inflammatory process going on in the tearing down of the bone to remove the temporary teeth and building up the process for the permanent teeth. So irregular is this action that in many mouths there is a continuous inflammation through the entire process on both jaws. This is noticeable in neurotics and degenerates and in those children whose vitality is low and who are poorly nourished. The bone is porous and loosely put together. The severity of interstitial gingivitis during the eruption of both sets of teeth and after all of the second teeth are in the mouth will depend upon the nervous system and blood supply. It must not be lost sight of, that, after all of the second set of teeth are in the mouth, there is a restlessness existing among them till all are securely located in their respective localities, although they may not be in their proper relations to each other. As long as this restlessness continues, and it may persist until middle life, interstitial gingivitis is always present. The irri-

tation set up by the advancing teeth causes inflammation in the gums and alveolar process. A visit to schools for defective children and an examination of their mouths will convince any one of the truth of this statement.

CHANGES IN FUNCTION.

Change in function has been discussed in a general way in previous chapters. It is necessary, however, to state here that owing to use and disuse of structures and environment, the function of mastication is gradually being reduced to a minimum. On this account and the gradual reduction of the size of the jaws and straight crowned teeth, the alveolar process, has, in most persons, changed its shape and has become narrow, long and thin. This change in shape and loss in blood and nerve supply makes the process about one or more teeth exceedingly susceptible to disease and destruction on account of loss of function.

TARTAR.

Tartar is the excess of lime salts in the blood excreted through the salivary glands. It remains soluble in the saliva until it reaches the mouth cavity when it precipitates. Just how precipitation is accomplished has not been proven beyond a doubt. One theory is that the action of ammonia exhaled from the lungs is the greatest factor.

Kirk says,[2] "As the saliva contains carbon dioxid in solution it has been assumed with some justification, that the escape of the carbon dioxid which was the solvent of the calcium carbonate and tricalcic phosphate, causes a precipitation of those salts in the presence of the colloid mucin, in combination with which it deposits as tartar upon the teeth."

H. H. Bouchard[3] proposed an explanation for the formation of salivary tartar deserving of consideration, viz., "that, inasmuch as fermentative processes in the oral cavity give rise to acids, and particularly lactic acid, these acids cause precipitation of the mucin of the saliva as a coagulum which entangles in its structure calcic phosphate and carbonate, and this mass by

---

2 Kirk. American Text-Book of Operative Dentistry, page 480.

3 See Origin of Salivary Calculus by Henry H. Bouchard, Dental Cosmos. 1895, vol. XXVII, page 821. Also Varieties of Dental Calculi, by the same author, Dental Cosmos, 1898, vol. XL, page 1.

gradual condensation increases in density to the extent of forming the coherent deposit known as tartar."

Tartar accumulations vary in quantity as well as in character in each individual and even in the same mouth. In some the greatest amount is found upon the molar teeth in opposition to the parotids whose function is apparently to keep the mouth moist. These glands constantly discharge small quantities of saliva without food stimulation. Owing to its special function, the sub-maxillary gland is stimulated by tasteful substances, hence probably does not send out as much lime salts. The sublingual glands next to the parotids discharge the greatest amount of calcic salts, which accounts for the quantity of deposit found on the posterior surface of the inferior incisors. The tartar will accumulate so rapidly in some mouths as to completely cover a tooth or teeth in a short time. In some autotoxic states there is always excess of tartar deposit. In a patient in whom malnutrition was most pronounced, deposits of tartar would collect so rapidly as to cover all the teeth in from four to six weeks. The teeth were of a degenerate type in that they had no enamel.

In tartar analysis there is also great variation, that deposited near Steno's duct having the greater per cent of lime carbonate while that from the lower incisors has the greater per cent of lime phosphate.

Tartar may be black, deep brown, green or yellow. In tobacco users the deposit is usually black, presumably stained from nicotine. Other agents will stain tartar as well. This is finely exampled in betel-nut chewers where "the rapid accumulation of large, dense deposits of tartar which at first are red, then finally become a dark, chestnut brown or black."[4] Nodules of tartar which cling with such persistency to the roots of the teeth are usually of a greenish or dark brown color and of great density. Tartar is not uncommon in domestic animals and the wild in captivity. I have no means of ascertaining whether wild animals running at large are thus affected.

If the deposits of tartar are allowed to remain upon the teeth, they are added to day by day. Sooner or later, they set

---

[4] Kirk. American Text-Book of Operative Dentistry.

up irritation and inflammation in the gums, in the peridental membrane and in the alveolar process, with resulting interstitial gingivitis, pyorrhœa alveolaris and finally exfoliation of the tooth or teeth.

IRREGULAR TEETH.

Every one is familiar with the fact that those persons who possess small jaws and irregular teeth are more subject to interstitial gingivitis than those persons possessing normal jaws and teeth. This is due first to the fact that those persons possessing irregular teeth have unstable nervous systems. Second, the irregularity of the teeth brings the roots closer together, making the alveolar process thin between the roots, thus reducing the blood and nerve supply, and thus reducing resistance. The gums are inflamed from want of cleanliness and proper brushing and the function of proper mastication is lost. The gums thicken because engorged with blood and stasis takes place, resulting in irritation.

UNCLEANLINESS.

One of the most fruitful sources of interstitial gingivitis is uncleanliness. Food and tartar collect on the teeth at the gingival border setting up irritation and inflammation. A want of proper measures for cleaning the teeth and gum margins after each meal soon allows the accumulation to irritate the gums which become inflamed and the food works its way along the roots of the teeth. The gums become inflamed, swollen and detached from the necks of the teeth. This condition allows more food and filth to collect which decomposes and forms lactic acid. This in turn also irritates the gums and further inflammation follows.

IRRITATIONS DUE TO MODERN DENTISTRY.

In my researches on interstitial gingivitis many years ago,[5] I called the attention of the profession to the fact that modern dentistry was producing more inflammation of the gums, peridental membrane and alveolar process than any one cause. These irritations were most noticeable at that early period about

[5] Talbot. The Dental Cosmos, Nov. 1886.

those teeth which had been "immediately separated for filling," so commonly practiced by the older members of our profession before the rubber dam. A piece of orangewood made V-shaped, with the base resting upon the gum margin, was forcibly driven between the teeth for the purpose of obtaining plenty of room for immediate filling and also to press forcibly against the gums and alveolar process to prevent the gums from weeping, thus keeping the cavity dry. In the majority of cases so treated, interstitial gingivitis was set up which in time became chronic.

The inflammation continued and followed the root upon one side oftentimes to the apex. The alveolar process became destroyed on the side of the inflammation and not infrequently pus infection would follow. The tooth would sometimes rotate upon itself or move in one direction or the other out of its position. The most excruciating pain accompanied the malleting of these wedges into place.

The modern method of rapid wedging with separators is only a modification of this barbarous method of procedure and is frequently the source of interstitial gingivitis and later pyorrhœa alveolaris.

Wedging teeth by any method sets up inflammation which may or may not be restored to normal. In any event, if poisons circulate in the blood later in life, such irritated or inflammatory localities, owing to the transitory nature of the structures, are the first to be affected by chronic interstitial gingivitis, since these structures have already been previously involved in structural changes.

CONDENSING GOLD WITH THE MALLET.

The excessive use of the mallet which was necessary to build out teeth in those early days assisted greatly in producing inflammation of the peridental membrane and alveolar process in connection with the rapid wedging. I recall a number of instances in my early practice where interstitial gingivitis and absorption of the alveolar process occured in neurotic children, the result of malleting in large gold fillings. These cavities were usually located in first permanent molars and sometimes in the

bicuspid. Absorption of the alveolar process took place, the teeth became loose and dropped out or were extracted.

At the present time malleting in large gold fillings in children's teeth, especially in neurotic, syphilitic, rachitic and similar children should be avoided on this account. The nerve strain, under such barbarous treatment, is very great and the boy or girl suffering from such constitutional conditions should be exempt from such procedure.

GOLD CROWNS.

When gold crowns were first introduced about 1880, the method of attachment was to drive the cutting edge of the gold to the alveolar process and disastrous results immediately followed. Inflammation set in and destruction of the alveolar process, with exfoliation of the tooth, soon followed. Since that time we have learned that gold crowns extending under the free margin of the gums will set up acute inflammation which afterwards becomes chronic and destruction of the alveolar process is sure to follow.

BRIDGEWORK.

When a bridge is placed upon two or more roots the function of those teeth has been destroyed. The teeth, owing to the elasticity of the peridental membrane, are allowed to yield slightly when pressure is applied in mastication. When they are bridged, they become rigid and the force of the impact in mastication irritates the membrane which together with autointoxication sets up further irritation and inflammation and absorption of the bone takes place. The collection of filth under bridgework is also a fruitful source of inflammation. Particles of food find lodgement under bridges and can not be dislodged by either tooth brush or toothpick.

DEVITALIZING THE PULPS OF THE TEETH.

Destroying the pulps of the teeth and filling the roots throw extra work upon the peridental membrane. This extra work changes the normal function of the tissues. Poisons circulating in the blood cause an inflammatory process to first attack such teeth and rapid inflammation and absorption occur.

CAVITIES IN TEETH AND OVERLAPPING FILLINGS.

Cavities at the margin of the gums and fillings with rough edges irritate the soft tissues and cause inflammation followed by absorption.

LIGATURES AND CLAMPS.

Ligatures and clamps for holding the rubber dam not infrequently irritate the gums and set up inflammation.

ARTIFICIAL DENTURES.

Partial plates of any description for artificial dentures or regulating plates act as foreign bodies against the mucous membrane and gums, producing irritation by heat, by the accumulation of foreign substances beneath them and the edge of the plate will irritate the gums about the teeth, thus setting up inflammation.

INSTRUMENTS.

Not infrequently instruments used in excavating cavities, finishing fillings, etc., will irritate the gum tissue which will later set up inflammation, absorption and finally destruction of the process.

EXCESSIVE BRUSHING.

It is not uncommon to find teeth located on the external surface of the alveolar process. This occurs in those patients in whom the jaws are small for the long diameter of the teeth. The teeth in erupting will force their way into place and when in their normal condition the dental arch is located upon the outer border of the alveolar process.

In such patients the bone over the roots of the teeth on the outside is often as thin as writing paper, while it is very thick upon the palatine and lingual side of the jaw. It is not uncommon to find the cuspid tooth located toward the outer side of the alveolar process while all the other teeth are in their proper places. The nerve and blood supply is almost nil, resistance or restoration is out of the question.

Under such conditions too great stimulation by the tooth brush causes inflammation and absorption of the outer plate of bone, which in time will expose the roots of the teeth.

PICKING THE TEETH.

The constant use of the toothpick, irritating the gum margin, is a fruitful source of interstitial gingivitis. This simple procedure is a spendid illustration of the effect of slight irritation producing inflammation of the gum which later becomes chronic with eventual destruction of the alveolar process.

REGULATING TEETH.

One of the most prolific sources of interstitial gingivitis is the regulation of teeth. When we consider the nature of the alveolar process and what it is obliged to undergo by the time the permanent teeth have erupted and the sensitive condition in which it is placed as a transitory structure and end organ, after the permanent teeth are in place, together with the unstable nervous system of the patient, as well as the age at which this operation must be performed, the wonder is that after the operation is complete there is any process left.

The pressure necessary to move the teeth sets up an inflammation to produce the entire destruction of bone in line of pressure. A third set of teeth in our present phylogenic development is unnecessary and therefore nature is ill prepared, especially in neurotic children with unstable nervous systems, to again build up the alveolar process about the teeth. The degree to which the alveolar process will be restored will depend upon the condition of the nervous system, the blood and the age of the patient. If the patient is poorly nourished so that material (lime salts) is insufficient or the nerve supply to the part is unstable or the patient has obtained his growth, the process is liable to be deficient in structure.

This deficiency in structure is easily demonstrable, in persons of middle age or of later life who have had teeth extracted, and a part of the alveolar process has come away. By frequent observation it will be seen that the process is not restored although the periosteum is still present. Again after the extraction of a tooth, although the alveolar process remains normal about the adjoining teeth, a slight absorption of the edges of the alveolus about the cavity will take place. The gums heal over the wound. By the use of a broken excavator, sharpened in a direct line with

the shaft at the point, this may be readily passed through the soft fibrous tissue of the once root cavity. Years after the extraction, fibrous tissue without lime salts, is present. The same is true when abscesses have formed. The fibrous tissue is restored but not the bone substance. Regulating teeth should be performed before the patient has obtained his growth and with as little movement of the teeth as possible to obtain fairly good results. The indiscriminate spreading of the dental arch without extraction should be discouraged. The excessive inflammation set up throughout the entire alveolar process for the purpose of enlarging the dental arch and thus bringing all teeth into line is liable to endanger the restorative process.

I have a record of forty-two patients in whom the teeth remained loose from a want of deposition of bone cells to hold them in position and in whom the roots of the teeth were exposed to a greater or lesser extent.[6] Interstitial gingivitis is always present and occasionally pyorrhœa alveolaris. The esthetic effect of the large dental arches associated with a small face is not in harmony with good judgment.

The nerve strain, the inartistic appearance and excessive inflammation should be reduced to a minimum by adopting such measures as are necessary to perform the operation as quickly as possible with as little nerve strain and with the least amount of work, to avoid interstitial gingivitis.

The removal of bone in front of the advancing tooth or teeth with a burr (as I have recommended) will save time, prevent undue inflammation, pain, nerve strain, and absorption.

PLANTATION OF TEETH.

Plantation of teeth consists of two methods. One the replacing of a tooth into the cavity from which it has been accidentally removed or a similar tooth has been taken from the mouth of another person, while the other is the insertion of a foreign tooth either into an enlarged natural socket or into an

---

[6] A young woman nineteen years of age called at my office for advice in regard to the restoration of the alveolar process about the six anterior inferior teeth. The bone had been destroyed in regulating. Retaining bands had been in place about two years. The operator was afraid to remove the bands for fear that the teeth would drop out. It was impossible to restore the process.

artificial alveolus which has been made for its reception. The replantation of a tooth which has been forcibly removed in a healthy growing child in most cases, if skillfully performed under aseptic conditions, will return to a normal condition without chronic inflammation.

On the other hand, from what we have learned from the unstable nature of the alveolar process and the many difficulties associated therewith, we hardly expect many favorable results by the enlargement of natural sockets or the formation of new sockets. By these operations the peridental membrane is destroyed in the one case and not present in the other. Interstitial gingivitis, with lacunar absorption of the root (which is a foreign body) takes place or absorption of the alveolar process by halisteresis ensues or both with the eventual loss of the tooth.

If the patient has obtained his growth, the chances of success are hardly to be expected. Especially is this true if the patient has autointoxication or other poisons in the blood or is subject to disease of any of the eliminating organs.

There are many other irritants, both constitutional and local, not mentioned by the author. Enough have been cited, however, to give the reader a fair idea of the influence of these irritants upon the alveolar process.

When inflammation is once established in the gums or alveolar process by local or constitutional conditions, it is usually progressive as far as the exfoliation of the tooth. If, however, the inflammation is circumscribed and does not extend entirely around a tooth which has one root, or, if only one root of a molar is involved, the inflammatory process, with absorption will have a limited area and extend only on one side of a single root or may involve only the one root of a molar tooth. This inflammation and absorption will progress to the end of the root. In other words, the disease is progressive after it has become established although treatment both local and constitutional may to a certain limit retard its progress. Illustrations of this may be recalled in those teeth which have been violently wedged apart to obtain room for filling and the inflammation extends along one side of a tooth, or, when gold crowns have been carried under the gums

and irritation has set up uniformly around the tooth, or, in teeth which have too much pressure in mastication, or, in those teeth for which there are no opposing teeth. The progressive nature of inflammation and absorption is due to the endo-transitory nature of the process.

CHAPTER XVIII.

CONSTITUTIONAL CAUSES OF INTERSTITIAL GINGIVITIS.

Pathological changes in the structure and function of the human body, in a more or less severe form, are due to constitutional affections. Sometimes they are the result of local diseases of certain organs, or the disease may have general characteristics from the first and still may affect certain individual organs, hence in this way secondary diseases are developed. This has been particularly borne out in trauma, intoxications, contagious infections and some tumor-like forms.

Diseases are designated as acute and chronic. Acute diseases are those of short duration. The acute stage may be terminated promptly in recovery or death or it may be prolonged in the chronic period. Disease may occur with or without reducing the tonicity of the body as a whole or a part.

Fever is a general metabolic disturbance, characteristic of many acute infections and autointoxications. The most important indication is a rise in the temperature of the body. The superficial temperature of a man normally varies considerably but the internal degree of heat is nearly constant. According to the researches of Jurgensen, Ziemssen and Krabler[1] the minimum internal degree of heat is during the early morning hours and the maximum is reached at about five o'clock in the afternoon.

In pyrexia, along with an increased production of heat, there is also an increase of nitrogenous metabolic products excreted in the urine. In regard to heat the amount thrown off varies; thus in the early stages of fever when the internal temperature is increasing, the surface temperature is below normal with contracted vessels and the amount of heat dissipation is lessened. Since the skin produces but a small amount of heat, its warmth is dependent upon the heat brought from the interior of the body,

---

[1] Thoma, Text-Book of General Pathology.

so if the skin vessels contract, the amount of blood that flows to the surface is lessened and the temperature of the skin falls.

The lessened amount of heat thrown off together with the greater production causes a rise within the body, although aside from a slight feeling of cold, there may be no apparent change in temperature. But if there is marked contraction of the skin vessels, the chilliness is more pronounced, so much that there may be violent shivering of the trunk, limbs and chattering of the teeth. The violent contraction of the skin muscles produces heat. When the fever is at its highest, the contracted cutaneous vessels yield, the skin becomes abnormally dry and hot, while the internal temperature remains high also. When the fever subsides, the temperature falls and the skin becomes moist, or there may be an excessive amount of sweat.

Other symptoms of pyrexia aside from temperature rise are malaise, headache, thirst, rapid pulse and respiration, digestive disturbance and decrease in the amount of urine secreted and passed with abnormal urinary acidity and oftentimes indican. In digestive disturbance, primarily, there is loss of appetite, excessive thirst, the salivary secretions are restricted and the entire alimentary tract becomes so affected as to prevent normal absorption from the stomach and intestine. The digestive and metabolic disturbance may be less than is usually assumed. Experiments show that in typhoid the digestion, absorption and utilization of food may be complete.

In a search for the connecting etiology of these fever symptoms we must first take into consideration the infections and intoxications from which they are derived. Disease producing germs, by their poisons when they enter the blood, cause fever. It may be that many substances produced by the metabolism of the human body have similar action. There are also poisonous agents not derived from microbes which cause temperature rise.

Fever appears to consist of two sets of symptoms, toxemia and pyrexia. The first are due to the direct action on the nervous system while the second constitute a reaction on the part of the system which tends at least to neutralize the effects of the toxemia. According to the researches of Vaughn, the poisonous substance is a derivative of the albumin molecule resulting from

a splitting such as occurs in digestion or in the destruction of bacteria by the blood serum (bacteriolysis). Thus certain vegetable proteins, like recin, peptones, and the results of the splitting of bacterial proteins excite fever. Metallic and alkaloidal poisons do not, as a rule, cause fever except by provoking inflammation which results in secondary bacterial infection. When these poisons are taken into the blood stream, a general faulty metabolism results which gives rise to the symptoms just mentioned. In regard to the pyrexial rise of temperature, particularly, it must be conceded that the poisons circulating in the blood have a disturbing influence on the structures of the central nervous system as well as on the vasomotor system of the peripheral nerves. The loss of heat, through the breath, is also controlled by the central nervous system which acts on the frequency and depth of respiration.

The normal heat of the body is produced by every organ in its metabolism. The principal heat producing sources, however, are the heart, muscles and principal abdominal organs. The energy of the heart muscle, when it contracts is exhibited partly as heat and partly as mechanical work. The heat produced by the contraction of the heart is partly radiated into the surrounding tissues and partly carried away by the blood. In addition, the mechanical work of the heart is completely transformed into heat by friction against the vessel walls and internal friction within the blood stream. The muscles all generate heat by their action. The disturbance of assimilation, digestion and absorption of food occurs at the same time, during fever, with temperature rise. This in turn causes metabolic disturbances not only of abnormal products but also a breaking down of organic cells. This may occur in various organs and there may appear any of the cerebral disturbances, such as headache, confusions, dizziness; also rapid decay of the teeth, interstitial gingivitis; absorption of the alveolar process; diseases and spontaneous death of the pulps of the teeth; erosion; abrasion and discoloration. After fevers, loss of the hair and abnormalities in the nails usually make their appearance.

Degeneration of tissues which form the substance of the heart, liver, kidneys and other structures, are partly due to dis-

turbances of high pressure and partly due to the direct action of poisons circulating in the blood which have caused the fever. It would not be strange, therefore, that similar degenerations occur as a result of simple action of poisons in non-febrile acute diseases as well as in poisoning by the various inorganic and organic chemical substances.

Chronic diseases are those of long standing and are usually the result of a prolonged acute condition. Generally there is no rise of temperature though there are instances of pyrexial and apyrexial periods alternating.

In a general way, I have explained the cause of some chronic diseases, contagions, infections and intoxications, but there are some constitutional disorders, for example, chlorosis, leukæmia, rachitis, obesity, gout, diabetes, osteomalacia whose etiology is still vague, yet are manifested by disturbed metabolism. Pathologic conditions to which these diseases give rise are many but the two most common are atrophy and malnutrition. One of the best examples of atrophy in the human body is the alveolar process. Atrophy as applied to the tissues and organs of the body is somewhat different in its action on the alveolar process. In all the other tissues and organs, atrophy means a gradual wasting away of structure. While the same condition takes place in the alveolar process, in addition to the wasting away, there is total destruction, owing to the fact that the process is a doubly transitory structure and an end organ. In the mouths of the congenital deaf, dumb, blind, feeble-minded and delinquent children, osteomalacia attacks the alveolar process before the osseous system has reached its growth. Here, as a consequence of trophic change, metabolic action and premature senility, osteomalacia may occur in connection with the first set of teeth at two years or any period thereafter. When this condition takes place early in life, I have called it "juvenile osteomalacia," late in life "senile."[2]

It is those organs of special function which become diseased and atrophy to the greatest extent; thus in the liver, the cell; in the kidney, the secreting epithelial cells; in the heart, the muscle fibers; in the spleen, the pulp cells; in the subcutaneous tissue,

[2] Pathogeny of Osteomalacia or Senile Atrophy. The Dental Digest, August, 1903.

the fat cells; in the lungs, the stroma of connective tissue and elastic fibers, the blood vessels and epithelial lining; in the bones, including the alveolar process, the lamellæ; the skin becomes thin and loses its tonicity; the epidermis dry, cracked and scaly; the brain diminishes and the space is filled either by atrophy of the skull or fluid in the pia-arachnoid.

Cachexia is characteristic of organ degeneration of a chronic type. In this condition, amyloid degeneration is sometimes associated with albuminous and fatty degeneration of the liver, kidney, heart muscle, etc. The fatty tissue is lost. The epidermis, unlike the marasmic states, is smooth and moist. The blood composition becomes changed, in many instances producing capillary hemorrhage or edema. Then, too, the weight of the body and organs is reduced, showing that cachetic conditions are a general disturbance of metabolism. Aside from its chronic character and absence of temperature variability, there are many similar features to those of the pyrexial disturbances of metabolism.

ACID STATES.

Having considered the more severe constitutional disturbances in which fever is always present, we must now briefly consider those constitutional disturbances so fatal to the alveolar process and in which fever does not manifest itself.

One of the most common causes of irritation producing inflammation and absorption of the alveolar process is the acid condition of the system. In the human body certain changes are continually taking place. These changes take place in the fluids of the body and are both physiologic and chemic. These changes add to and take away tissues of the body and are alkaline or acid. They are called anabolic when the fluids are alkaline and the tissues are built up and katabolic when the fluids are acid and the tissues are broken down. The alkaline and acid states of the body may be ascertained by the examination of the excretions of the body. When the fluids of the body (except the gastric juice) have an excess of acids the saliva, mucus, perspiration and urine will be excessively acid. When the secretions of the

body are acid it indicates that there is a diminished alkalinity of the blood. This in turn leads to improper functioning and prevents proper nutrition and produces lowered vitality.

It is known that an acid excess in the system will hinder and often destroy the transmission of nerve impulses. Thus in acid states large areas of skin will be without sensation; the knee jerk is diminished or lost altogether. A continued acid condition of the system will cause nerve end degeneration in the pulps and fibrillæ of the teeth. The teeth will discolor, become brittle and the enamel and dentine will break off. In no part of the body does the excessive acidity manifest itself as in the alveolar process. Its endo-transitory nature makes it very susceptible to irritation through its nerve filaments and its end arteries, setting up irritation and inflammation. When the fluids of the body are acid the alveolar process and mucus excreted are, therefore, acid to a greater or less extent, nutrition is thus cut off and absorption of the process takes place. Thus in acid states, as well as in dental states, the alveolar process gradually absorbs away.

In dealing with the influence of buccal states on the constitution it must be remembered that when the eliminatory system is overstrained, especially when the poison-destroying function of the liver is deficient or impotent, the alveolar process and gums play a great part in elimination, whence come, for example, the "blue" gums of lead poisoning and the "green" gums of brass, as well as those from mercury, arsenic, potassium iodid, bromid, etc. Matter thus eliminated is reabsorbed, enters into the chyle with digested products, and readily becomes toxic to the blood cells. That cachetic states, approximating pernicious anæmia, can thus be produced, is clearly evident. Were fecal anæmia existent before the gum and alveolar process changes were set up it would thus be greatly intensified. The toxemia producing this gum and alveolar process state would be greatly increased through the overstrain of oxidizing processes produced by reabsorption of eliminated products.

The toxins generated in the mouth readily pass into the general system. As a result, chronic indigestion with coexistent pigment spots, urticaria, etc., may occur. Pus toxins may thus

produce a sapremia mimicing typhoid, as pigment spots readily simulate the typhoid eruption. In buccal manifestations of constitutional disease the vicious circle of pathology peculiarly occurs.

The alveolar process may be affected at any period of life after the eruption of the first set of teeth but osteomalacia does not usually occur until the period between twenty-five and thirty-five. Before this the osseous system is in its constructive state and lime salts are being deposited rapidly. Later in life the constructive stage is complete and material sufficient only to repair waste is deposited. At the periods of stress metabolic changes are most active—during puberty and adolescence (fourteen to twenty-five), during the climacteric (forty to sixty), when uterine involution in women and prostatic involution in men occur and finally during senility (from sixty upwards), when the disease is always present to a greater or lesser degree. While in allied conditions men are most influenced, in this disorder the sexes seem to be affected about equally. Here the influence of pregnancy comes into play. Pregnancy disturbs the physiologic balance hitherto existing, especially along the line of assimilation and elimination. The well known dental effects of pregnancy (whose underlying cause affects the alveolar process) are due to this factor. This is purely a constitutional affection.

Among the causes are non-elimination of toxic substances, whether due to autointoxication, to bacterial action, or to metallic and vegetable drugs. Disorder or disease of any excretory organ (kidneys, bowels, skin or lungs) will produce the most marked effect, first upon the constitution of the blood, and second upon the alveolar process, with resultant osteomalacia.

The urine, as has been shown, contains each day in a normal individual sufficient toxins to cause death if not excreted. This condition is markedly increased after prolonged nervous explosions like those of epilepsy or hysteria. This was pointed out thirty years ago by Meynert, who demonstrated that the status epilepticus (condition of rapidly-recurring convulsions) was due to the accumulations of a proteid body in the system. The status epilepticus is preceded by a decrease of toxins in the

urine and succeeded by an increase. This is likewise true as to the influence of non-elimination by the other excretory organs (bowels, lungs and oral cavity), as well as to the non-exercise of its poison-destroying power by the liver. Non-elimination moreover interferes with ordinary digestive functions and hence increases its own extent. Another factor in autointoxication is production of toxic products in such quantity as to prevent destruction by organs like the liver and consequent elimination, since a product to be properly eliminated must be changed to a particular chemical type. Among the factors which affect both these elements of elimination is the power over growth and repair exercised by the nervous system. In part this influence is exerted through control of blood supply by the vasomotor nervous system, and in part by that direct control of the nervous system over tissue change which is known as its trophic function.

Both influences are affected by nerve strain. Sudden emotion may, as Bichat demonstrated decades ago, produce marked defects upon bile secretion and may occasion jaundice. Cases are far from infrequent in which emotions like jealousy produce a mimicry of gall-stone colic in neuropaths. Murchison, Christison and Thompson have traced attacks of biliary colic to jealousy. Other liver changes from sudden nervous disturbance, whether of mental type or not, are not rare. As mental impressions are communicated to the central nervous system purely through mechanical changes in the nerves, such influence must be purely material in operation. As the brain exercises a checking influence on the operations of the liver, these mental influences produce two effects. The mental shock increases the checking action of the central nervous system on the local ganglia of the liver, and destroys the checking action of the liver ganglia, and in consequence these go too fast, resulting in their exhaustion. Either of these conditions interferes with the poison-destroying action of the liver, and accumulation of waste products is the result.

What is true of the liver is true of the other organs. This is especially noticeable, as Tuke points out, in regard to the kidneys. The action of mental anxiety or suspense, in causing a

copious discharge of pale fluid, is familiar enough to all, especially to the medical student about to present himself for examination, the amount being in a pretty direct ratio to his fear of being plucked. The frequency of micturition may, however, arise from nervous irritability of the bladder without increase or even with diminished secretion. Still the action of the skin is usually checked, the extremities are cold, and the kidneys have to pump off the extra amount of fluid retained in the circulation. Elimination of the substance usually separated from the blood is diminished as compared with the aqueous character of the whole secretion. The odor may be affected by the emotions in man as in animals. Prout is of the opinion that mental anxiety will produce not only non-elimination but also change in the chemical character, as indicated by odor and otherwise. Disturbances in the medulla produce, as Claude Bernard long ago showed, a markedly pale, excessive urine. These disturbances often arise from intellectual strain or emotional shock. The influence of emotional states on secreting processes, and thereby indirectly upon autointoxication states, is illustrated in the fact long ago pointed out by Tuke that pleasurable emotions increase the amount of gastric juices secreted, the opposite effect being produced by depressing passions. Beaumont found in a case of gastric fistula that anger or other severe emotions caused the gastric inner or mucous coat to become morbidly red, dry and irritable, occasioning at the same time a temporary fit of indigestion.

The influence of fear and anxiety on the bowels is as well marked as that upon the bladder and kidneys. Apart from muscular action, defecation may become urgent or occur involuntarily from various causes. The increased secretion from the intestinal canal may occur from fear and in some cases from the altered character of the secretion itself. While in this respect the influence of fear may be inconvenient in man, it naturally assists escape in some animals, as the skunk.

Emotions powerfully excite, modify or altogether suspend, as Tuke has shown, the organic functions. This influence is transmitted not only through the vasomotor nerves but through nerves in close relation to nutrition and secretion. When the

excitement is of peripheral origin in sensory or afferent nerves, it excites their function by reflex action, so that as emotion arises it may excite the central nuclei of such afferent nerves, and this stimulus be reflected upon the efferent nerves, or it may act directly through the latter. Pleasurable emotions tend to excite the processes of nutrition, hence the excitement of certain feelings may, if definitely directed, restore healthy action to an affected part. Violent emotions modify nutrition. Various forms of disease originating in perverted or defective nutrition may be caused primarily by emotional disturbance. Emotions, by causing a larger amount of blood to be transmitted to a gland, increase sensibility and warmth and stimulate its function or directly excite the process by their influence or nerves supplying the glands. Painful emotions may modify the quality (i. e. the relative proportion of the constituents) of the secretions.

Imperfect elimination of effete matter from the lungs is a fruitful source of autointoxication. The more marked forms are those of tuberculosis, in which there is great debility and in which there is greater waste than repair. Self-poisoning is continually going on and will continue until death. The chest capacity for the inhalation of pure air is almost nil, hence the blood is improperly oxygenated and soon ceases to convey nutriment to the tissues. Eight per cent of criminals who die of tuberculosis in prisons have undeveloped chest walls. Degeneracy therefore cuts quite a figure in the rôle of autointoxication. Degenerates with contracted chest walls are, however, more frequently found. Many undeveloped individuals in every walk of life for this reason have tuberculosis. People with undeveloped chest walls and chest capacity may not have tuberculosis and yet may suffer from autointoxication. Those who have had pneumonia with adhesion; and who are thus unable to oxygenate the blood, are subject to this disease. Asthmatics and hay-fever patients suffer from autointoxication and alveolar absorption. When the skin is overstrained as to excretion through kidney and bowel overstrain, the lungs are forced to take on increased work with imperfect oxygenation as a result. This is noticed in the odor of the breath in Bright's disease and in the air-hunger of diabetes, etc. In nerve-strain states and

in the condition described by Albu not only do excretory organs suffer but the secretions of those glands like salivary and buccal glands are so altered as to become irritants. These excretory conditions not only result upon autointoxication states but are modified by trophic nerve function alterations. By trophic changes are meant such tissue alterations as occur in morbid conditions from disordered function of the centers of nutrition. Peripheral as well as central may be involved. The well known law of Wallerian degeneration of nerve fibers is an illustration, the posterior ganglion acting as a trophic center for the fibers of the posterior root in the cord itself. Trophic action may therefore be peripheral, though in extensive changes as a rule central (cerebral or spinal) origin should be looked for.

The constitutional result of acute and chronic infection and contagions is apt to be an autointoxication plus the action of the germ toxin. All the exanthemata have at times been followed by wasting or necrosis of the alveolus. Here the condition is notably symmetric and accompanied by disorders of the osseous system elsewhere. The same is true of grippe and tuberculosis. The well-marked disorder known as Riggs' disease has been charged by Pierce, Kirk, Rhein, Robin and Magitot to the direct influence of an arthritic state (gouty and rheumatic) and regarded as a special type of arthritic manifestation. The alveolus is clearly vulnerable to the toxins of many infections. It is likewise quickly affected by some autotoxic influences from disordered metabolism. Its vital resistance to these agencies is less than that of other tissues. It is the earliest sacrifice when these or any toxins disturb the harmony of the organism.

A cause other than the actions of toxins exists for implication of these parts. Whenever tissue waste, whether local or general, exceeds repair there is trophic change. This latter depends directly upon disordered local or general nervous functions. Trophic alterations from the first cause appear in growth disorders of the nails and loss of hair (alopecia) after fevers, the most familiar obvious examples of this pathologic process. Of the other type are localized atrophies where the direct inter-

vention of toxins can be excluded. The alveolus is liable to the first form of trophic deterioration. The influence of acute diseases upon the alveolus is probably thus exerted in many cases rather than by direct infection. Where no cause has been ascertained examination directed to this factor would probably reveal it. The general failure of the trophic centers after the prime of life (in senile states), which is attended with loss of teeth and wasting of the alveoli, is the most obvious instance of trophic failure affecting the part. Even simple anaemia may thus give rise to alveolar wasting.

Another constitutional disorder in which the alveolus is early affected is diabetes. The exact pathology of this is un-

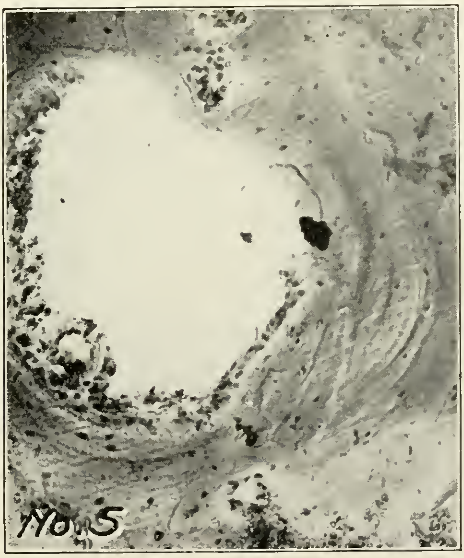


FIG. 75.—ABSORPTION BY HALISTERESIS. THREE SMALL ARTERIES ARE SEEN. TWO IN THE UPPER PART OF THE ILLUSTRATION WITH ROUND CELL INFLAMMATION JUST BEGINNING, WHILE ANOTHER SMALL ARTERY IS LOCATED AT THE LOWER BORDER OF THE LARGE ABSORBED AREA.

certain, but in many cases at least it is largely dependent on disordered action of the central nerve system. Renal disease is another common condition which tests the vulnerability of the alveoli. An ideally normal kidney is probably rare, but only when its abnormalities pass beyond a certain point can it be called diseased. In the less advanced conditions that have passed the line of morbidity, alveolar implication is often very

marked. This may be one cause of the unusual frequency in the insane, who are especially liable (as Bondurant and others have shown) to suffer from renal disease. They are very liable likewise to autointoxications and trophic disorders as well, since the balance of the nervous system has been upset. Some (the paretic and organic dements) exhibit especial tendencies to trophoneurotic disturbances affecting the teeth. In states of depression and stupor, circulatory disturbances predispose to these.

The constitutional results of acute and chronic infections are apt to include autointoxication in addition to the action of the toxins of their germs. The eruptive fevers, especially scarlatina or measles, have been long known to be followed by wasting or necrosis of the alveoli. Here the condition is notably symmetrical and unaccompanied by exfoliation or necrosis of the osseous system elsewhere. Tuberculosis does not spare the alveolar process.

The more marked forms of constitutional disorders (typhoid fever, pneumonia, tuberculosis, syphilis, indigestion and pregnancy, etc.) produce intense results.

The second form of trophic failure in the alveolus is less prominent, since it generally coexists with overshadowing disturbance elsewhere which it creates to a certain extent. Cruveilier noticed its occurrence associated with simple paraplegia, regarding it as of nervous causation. In facial hemiatrophy local wasting of the alveolus has appeared before the disorder has involved the jaws generally. This is sometimes due to a local cause, but its occurrence and association with other neurotrophic symptoms are suggestive.

We have seen that the action of the heart plays quite a rôle in the constitutional diseases of the body. Constitutional diseases affecting the heart and diseases of the heart itself cause excessive and diminished action of this organ. This excessive and diminished action causes changes in the flow of the blood in the peripheral capillaries. Those organs first involved in this change of heart pressure are the end organs of the body. The alveolar process, therefore, being the most sensitive end

organ, because of its transitory nature and its bony substance is one of the first to be involved in autointoxication.

In intestinal putrefaction, hepatic and renal insufficiency and drug poisoning as well as in other diseases, the heart becomes enlarged and a high blood pressure is developed. Dilation of the arteries occurs, especially in those of end organs, resulting in arterio-sclerosis, which is present in every case.

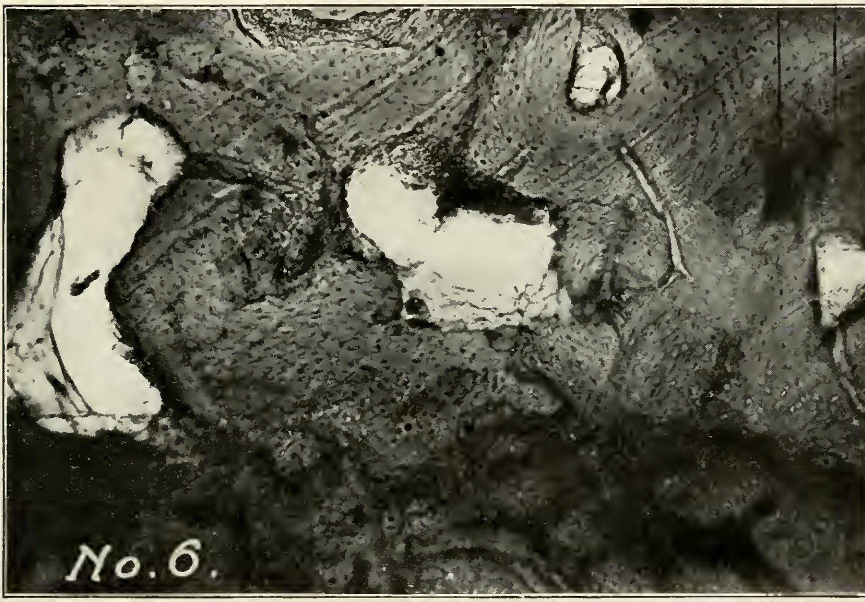


FIG. 76.—SHOWS BONE ABSORPTION BY HALISTERESIS, AND VOLKMAN CANAL ABSORPTION.

To ascertain the blood pressure in patients suffering with interstitial gingivitis I used Cook's modification of the Riva Rocci sphygmomanometer, this instrument being best adapted for my convenience and exceedingly simple. The armlet used was sold with the instrument and consists of a rubber bag 4½ by 40 cm. The patients ranged from twenty-seven to sixty-seven years of age. With this instrument the normal adult female arterial blood pressure is 115 to 125 mm.; adult male, 125 to 135 mm.

In twenty-six females there were three who ranged between 115 mm. Hg. and 125 mm. Hg. and therefore normal. Three

ranged below 115 mm. Hg., and twenty from 133 mm. Hg. to 180 mm. Hg.

In twenty-four males there were eight who ranged between 125 mm. Hg. and 135 mm. Hg. and therefore normal. Three ranged below 125 mm. Hg., and thirteen from 133 mm. Hg. and 160 mm. Hg.

When we consider that thirteen of these patients were under forty-five years of age, the high blood pressure is remarkable.

I have been unable to demonstrate whether the interstitial gingivitis is accelerated directly because of the poisons circulat-

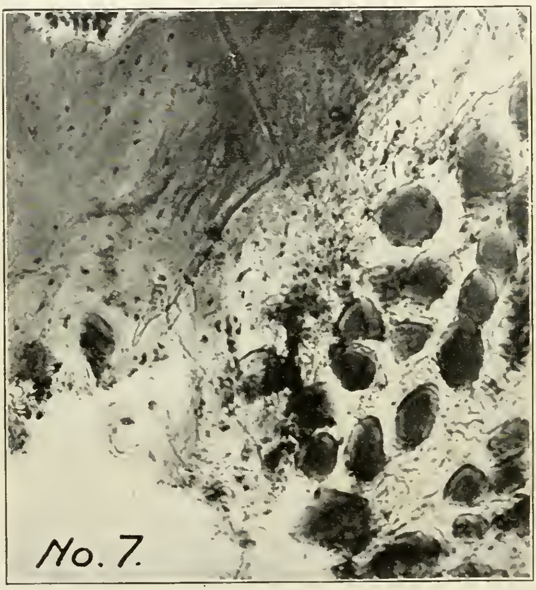


Fig. 77.—Illustrates Lacunae or Osteoclast Absorption.

ing in the blood vessels, causing high blood pressure by their action upon the heart, or because of their action upon the vasomotor nerve governing the heart or blood vessels, or both. The effect of the toxins and extra blood pressure is to set up irritation and inflammation of the outer surfaces of the Haversian canals, producing halisteresis in the vessels of Von Ebner, producing Volkmanns' perforating canal absorption and setting the osteoclasts at work, all producing absorption of the alveolar process.

The question arises, which end organ is the most susceptible and first involved in autointoxication? When a man visits the physician for treatment one of the first questions asked is, "What is your occupation?" If the man replies that he is working in drugs, metals or mines, the physician examines his patient's gums to note if his system be saturated with poisons. If a physician is treating a patient for lues, the drug is administered until the "gums are touched," which is the only indication his patient is under the influence. One of the most marked symptoms of scurvy is the inflammatory condition of the gums and alveolar process, which are always taken into consideration in diagnosis.

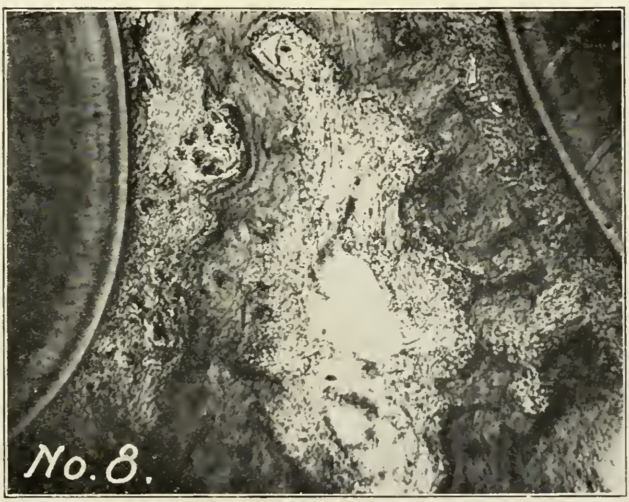


Fig. 78.—Lower Portion Showing Absorption of the Alveolar Process Below the Roots of the Teeth.

Physicians agree that the arteries in such end organs as the kidneys, brain and retina dilate under blood pressure. The arteries ramifying bone structure, dilate only imperfectly, if at all. Arteries entering transitory bone structures gradually undergo pathologic changes. After the individual has obtained his growth these arteries certainly are more susceptible to toxin, poison and blood pressure than those in the kidney, brain or retina. I have demonstrated these pathologic changes in the alveolar process many times. The toxic products circulating in the blood affect the heart and cause a high blood pressure.

High blood pressure, together with toxic products circulating in the blood, set up inflammation in the alveolar process and gingival border. In the alveolar process, first, because the arteries in the soft gum tissues, under high blood pressure, can and do expand and the tissues recover as soon as the cause is removed, but the arteries running tortuously through the bone cannot expand to any appreciable extent, and the blood pressure and toxic products cause inflammation and absorption of bone tissue without restoration. Hence the term "interstitial gingivitis" (deep-seated inflammation in the alveolar process). Cardio vascular, nervous, hepatic and renal diseases, as related to interstitial gingivitis, are therefore due to the same cause. In relieving or removing the cause of interstitial gingivitis the other symptoms and diseases are relieved, and vice versa.

Figure 75 illustrates a large area of absorption with destruction of the fibrous tissue to a larger extent. Around the border is seen a small amount of inflamed fibrous tissue. An artery, once an Haversian canal, is also seen. About the large area are also seen three Haversian canals with the inflammatory process just beginning.

Figure 76 shows four centers of absorption at Haversian canals. Through the picture may be seen dark lines running in all directions. These are vessels of Von Ebner, through which Volkmann's canal absorption takes place. A beautiful illustration of this is the canal running from one large area of absorption to the other.

Figure 77 shows the third form of bone absorption—lacunæ or osteoclast absorption. Here a large area of bone is destroyed by these large cells.

Figure 78 is a low power, showing the distribution of the alveolar process between the roots of two teeth. Very little of the bone remains. When the trabeculæ or fibrous tissue is destroyed in large areas, and especially in transitory structures, it is rarely restored.

CHAPTER XIX.

CLIMATIC INFLUENCES IN INTERSTITIAL GINGIVITIS.

One of the simplest forms of constitutional disturbances which produce interstitial gingivitis is that of climatic changes. The effect of climate which includes heat, cold, moisture, dryness is generally recognized by physicians as having much to do with the action of disease upon the system. To such an extent has this subject been impressed on the profession that the late Dr. N. S. Davis of Chicago, many years ago organized a separate section in the American Medical Association on Climatology, while every medical congress has its section on Tropical Medicine. If this subject is of so much importance in the cause and treatment of disease, especially in its influences upon the excretory organs, how much more important must it be in relation to interstitial gingivitis, since the alveolar process, being a doubly transitory structure and an end organ, is always the first affected by sudden and prolonged changes.

Experiments have shown that cold acts as a stimulant and increases the amount of carbonic acid exhaled, while experiments made by Dfluger and Marcet also show that a similar increase from heat would produce the same result. Both cold and heat then to a certain point act as stimulants.

The judicious change in climate, that is, moving in winter to warmer climates and vice versa, taking into consideration moisture, light, rarification or condensation of air and the increase by the variations in the manner of life and hygiene may prove beneficial or disastrous according as the organs respond to the changed environment.

In low temperature, the body loses more heat and the loss must be supplied. The older the individual the more marked is the effect of excessive cold and heat. The effects upon the individual are the same as in high temperatures, in part through local injury and death of tissue, in part through refrigeration of the entire body. Severe and lasting lowering of tempera-

ture causes tissue death; after mild chilling has occurred, as the result of tissue degeneration, thrombosis, hyperaemia and exudations which are relatively rich in leucocytes. A very short refrigeration at the freezing point is sufficient to produce degenerative changes which are quickly followed by regenerative proliferation on the part of the cells remaining uninjured. Tips of the extremities, nose, ears, fingers, feet and toes, are the most easily frozen because of their extreme distance from the heart.

According to Ziegler, "Besides the more severe forms of local or general lowering of the tissue temperature there may occur, as harmful pathogenic influences, mild, general or local chillings, the so-called *colds,* as the result of which disease-phenomena may manifest themselves partly at the seat of chilling, partly in organs in distant parts of the body. For example, after widespread refrigeration of the skin there may occur diarrhoea, catarrh of the respiratory tract, or disease of the kidneys; after local chilling of the skin, painful affections of the deep-seated muscles. The exact relation between these phenomena and the refrigeration is unknown (the oft repeated hypothesis that they are due to hyperæmia of the internal organs caused by the chilling of the body surface has not been proven), but there is no reason on this account to deny the existence of diseases caused by cold. Though many diseases formerly attributed to "catching cold" have been known to be of infectious origin, there yet remain a number of diseased conditions for which we know no other etiology than that of refrigeration. Conditions of the body in which the skin is hyperæmic and the perspiratory function active favor the taking of cold. Many individuals appear to possess a predisposition on the part of certain tissues to the effects of refrigeration; in one person certain muscles, in another the mucous membranes will be affected.

According to the view of many writers, refrigeration of the body increases the susceptibility to infection, so that, for example, the pathogenic bacteria which may be present in those cavities of the body accessible from without, may, after such refrigeration, be able to exert their injurious influences upon the tissues."

There is a great difference between radiated or sun heat and shade heat. Sunstroke is rare in the pure and comparatively dry air of high elevation. The same is true on the ocean. Shade heat, on the other hand, can be borne less easily. One can do hard work in the sun heat when one would rapidly become exhausted in shade heat at one-third the temperature. People bear heat very differently. The manner of living must necessarily be taken into consideration. It has been shown that Europeans who go to live in hot climates are injured by continuing the same kind and amount of food and stimulants as they are accustomed to use at home. Parkes says, "great heat in shade exerts a depressing influence lessening the great functions of digestion, respiration, sanguinification and directly or indirectly the formation and destruction of tissue."

Dr. G. D. Boak states as to Philippine climatic effects upon the teeth: "While the weather is by no means as hot as it is at times during the summer in the States, the average temperature for the islands is about 89° F. It is a continuous heat without invigorating change of seasons. This gradually saps vitality and enervates, producing the lassitude which is characteristic of the tropics. Enervation produces anæmia, with corresponding lessening of the resisting powers from the lower vitality, especially in those who have lived previously in temperate climates. Caries is frequent and progresses rapidly in this climate." This Dr. Boak attributes to the following causes: First, lowering of the vitality by a lessening of the resisting powers; second, acidity of the oral secretions.

Among important factors to be considered in connection with hygiene in the tropics are the questions of dietetics as well as the effects of moist and dry heat. The two last produce, as elsewhere shown, a neurasthenia with co-existing and complicating autointoxication. These two peculiarly affect the alveolar process. It is, therefore, not remarkable to find in a recent report by General Otis, the case of Walter Fitzgerald, Company C, twenty-ninth infantry, formerly of the Montana volunteers, cited. This twenty-three-year-old man had been in the Philippines for a year and seven months. He was one of the first volunteers to reach Manila after the naval battle. Nine-

teen months' life in the tropics on the usual army rations had resulted in the loss of nearly every tooth. While the climate undermines nutrition of the alveolar process, and tropical fevers have the same effect, improper diet increases the defect. In the case of Fitzgerald, the teeth dropped out one by one, as is commonly the case with Americans in the Philippines.

Soldiers going from a temperate climate to Cuba and the Philippines with change of food, had autointoxication and interstitial gingivitis.

In an examination of the soldiers and officers of two companies who had just returned from the Philippines, located at Fort Sheridan, Illinois, I obtained the following results: The total number examined was 127. American, 98; Irish, 12; German, 9; English, 3; Norwegian, 1; South American, 1; Danish, 1; Russian, 1; Cuban, 1. The ages ranged from 21 to 52. By ages the following data was obtained:

Age	Disease	Age	Disease	Age	Disease
21	8	32	7	43	1
22	5	33	1	44	1
23	17	34	2	45	none
24	12	35	none	46	1
25	15	36	1	47	1
26	9	37	7	48	none
27	4	38	1	49	none
28	8	39	3	50	1
29	11	40	none	51	none
30	5	41	none	52	1
31	4	42	1		

Total: 18 none; marked 36; medium 27; slight 46. Percentage: 14.1 none; 28.3 marked; 21.2 medium; 36.2 slight.

In studying these figures it will be noted the largest number of cases of interstitial gingivitis occurred between the ages of 21 and 30, the period of life at the constructive stage when the disease should not be present. Those over forty were nearly all officers who took better care of the mouth. It must be also noted these men lived most of the time in the open air. In the American army mostly young men are enlisted. It will be seen that the effect of climate and food is very severe.[1]

Even in moderate temperatures where changes of climate

[1] The Dental Summary, 1903.

have taken place, strong persons, after long exposure to such temperatures, undergo a certain degree of lassitude, diminution of appetite and impairment of functions of digestion, respiration, circulation and metabolism. On the other hand, it is also true that weak persons may gain in weight and the functions of the mind and body be much improved.

The sudden changes in temperature and atmospheric pressure, such as mountain climbing, balloon and aeroplane ascension, may cause great exhaustion, palpitation of the heart, irregular breathing, unconsciousness, and sometimes vomiting, with bleeding of the gums. It is claimed by some research workers that the capillaries of the lungs are unable to take up sufficient oxygen from the highly rarified air. According to the investigations of Schumburg and Zuntz it appears that a given amount of labor calls for a greater amount of oxygen in an increased elevation than in a lower level. It would seem, however, that the sudden changes from moderate to extreme cold or heat act upon the body in such a manner that the eliminating organs are unable to adjust themselves quickly to the new environment. The result of this is that autointoxication takes place and faulty metabolism is produced.

During the building of the Gornergrat Railway in Switzerland, it was found that at a height of three thousand meters, the capacity of the laborers was diminished to one-third. According to the researches of Egger, Miescher and others, a sojourn in high altitudes leads, after a short time, to an increase in the number of red cells and a greater hæmoglobin content in the blood.

Hafner[2] of Zurich has recently shown that "the engineers and workmen on the Jungfrau railway, obliged to remain a considerable time at altitudes of about 2,600 meters above the sea level, are liable to a disagreeable complaint. After eight or ten days they are seized with violent pains in several teeth on one side of the jaw, the gums and cheek on the same side becoming swollen. The teeth are very sensitive to pressure, so that mastication is extremely painful. These symptoms increase in

[2] Die Natur, 1900.

severity for three days and then gradually and entirely disappear. It seems to be purely a phenomenon of acclimatization. All new comers pass through the experience and the disorder never recurs." The influence of heat, of cold and of the barometric pressure shown in a lesser degree in "mountain fever" produce systemic disturbance of metabolism which, causing autointoxication, markedly affect the alveolar process, producing interstitial gingivitis.

The author has examined the mouths of the workmen on the Jungfrau, Gornergrat and Pillatus Railways at various times and can confirm the statements made in regard to the condition of the men as stated above. The quality of food, the unhygienic condition of the mouth and high altitude have acted severely upon the alveolar process, causing the teeth to loosen and drop out. While the pathology is similar to scurvy, the other constitutional symptoms, associated with scurvy, are not observable in these patients.

When the Government authorities made their report on the survey of the State of Minnesota many years ago, they made the claim that it was impossible for human beings to live the entire year in that state owing to the extreme cold in winter. It is now known that in the Northwest, owing to the extreme cold in winter, people who live in exceedingly hot rooms suffer from the extremes of cold and heat and are subject to more severe interstitial gingivitis than those in more moderate temperatures in the United States.

The results of these extremes in temperature produce many affections of the heart from a want of quick adjustment of the eliminating organs to the new environment. In high altitudes and cold climates, the skin contracts and elimination of the body waste is thrown upon the internal organs, and vice versa in warm climates.

The result is that in these sudden extremes, the functions of the body are slow in adjusting themselves to environment, nutrition is interfered with, vitality is lowered and the structures of the body are affected in the order of their sensibility to autointoxic states. The peripheral nerves are usually the first involved; then the arterial coats and the blood stream are inter-

fered with. Transitory structures and end organs thus receive the first impulses of faulty metabolism.

This sets up an inflammation in the capillaries of the alveolar process. No matter how short a time the cause may exist, if the inflammation is once set up, owing to its peculiar action on the alveolar process (even though the cause be removed), interstitial gingivitis becomes chronic and the destruction of tissue continues.

CHAPTER XX.

SCURVY IN INTERSTITIAL GINGIVITIS.

Scurvy is due to poor food and improper hygiene. Insufficient alternation of food, impure air, want of bodily exercise, ennui and uncleanliness combine to form the causes of this disease. Previous to the introduction of canned goods, sailors on long voyages, prisoners and others under confinement were subject to scurvy. Lunatics, idiots or persons who have had long illness, and find it difficult to regulate their diet, are now most prone to it. Anæmic convalescents from protracted fevers suffer from it. Bottle-fed babies and occasionally those fed at the breast on non-nourishing milk are prone to the disease.

In the British Arctic Expedition of 1875-76 over forty-eight per cent of the men suffered from scurvy. When the potato crop failed in Ireland, in 1846, scurvy became prevalent. In the Crimean war 23,000 cases occurred among the French troops alone. Scurvy contributed over fifteen per cent to the death rate in the late civil war. It occurs among the Klondyke miners.

Thomas Barlow found scurvy associated with rachitis. Sunderland found that rachitic diathesis was a very strong factor. Jacobi reports forty cases of scurvy and rachitis. Babies in good families brought up solely on the proprietary infant foods are prone to scurvy. They lose their appetites, become pallid, perspire freely, have diarrhœa, the mouth becomes sore, with inflamed mucous membrane and gums. Purpura and hemorrhages of mucous membrane are common with pain and swelling of the joints.

Just how far scurvy may be associated with extreme changes in climate in relation to interstitial gingivitis is an open question, but soldiers going from one climate to another, as exampled by American soldiers to Cuba and the Philippines, and British soldiers to South Africa and India, are more subject to scurvy

[1] Brit. Epidemiological Society, Feb. 19, 1904.

from improper food than native soldiers. Mayer Coplans [1] of the British Army Medical Service states, in regard to the South African War, that scurvy developed among the local population, in the concentration camps, fed upon Government rations which were of fairly good quality. The concentration of native women and children in camps containing about 5,000 persons, forty-eight per cent of children under twelve years of age contracted the disease. Among adults the women were to the men as three to one. Enclosed by barbed wire, the camps, though open and airy, were securely isolated. The conditions being identical the varying incidence of scurvy was remarkable. Between March, 1901, and January, 1903, with no cases at Standerton or Volksrust, there were one hundred Europeans and one native attacked. Among the soldiers at Standerton and the 22,000 European patients admitted to the hospital there was but one; among the natives in the service of the troops there were attacked 32 per cent of 400 muleteers, 22 per cent of those attached to the Hussars and the Royal Artillery, 87 per cent of the scavengers engaged in removing carcasses of animals, 17 per cent of the porters and about 50 per cent of the muleteers in the employ of the repatriation department.

The heaviest incidence of scurvy was after the close of the war and when all restriction on food had been removed. In fact, it had no relation whatever to the food but was almost everywhere directly in proportion to the neglect of cleanliness, of which the natives had not the most rudimentary notions, especially as regards the hygiene of the mouth. Even the outbreak among the burghers at Middleburg followed overcrowding and neglect of sanitation.

A. E. Wright, in discussing the subject, said filthy habits were not peculiar to the Kaffirs, and were not always accompanied by scurvy, which occurred in the nurseries of the rich and in nursing homes. Scurvy was essentially an acid intoxication, a reduction in the alkalinity of the blood which can be observed long before the grosser manifestations, alike in the adult and infantile forms. A large proportion of the troops returning from South Africa were scorbutic in the latent stage.

K. B. Goadby also, in a discussion of the subject, said he had not seen any scurvy at the Dreadnaught Hospital, but had met much pyorrhœa alveolaris, a disease endemic and occasionally epidemic in West and Central Africa, the Transkei, the Philippines and other places. This condition of the gums and the rapid recovery of the patients under antiseptic measures resembled that found in scurvy.

Coplans pointed out in reply that it required months for its development, for the members of the corps that suffered most were recruited in their homes in October and the corps was dissolved in December, the disease breaking out soon after their arrival in the camp. Recovery followed rapidly on purely local treatment in the way of buccal antiseptics without any attempt to influence the blood.

The term "scurvy" frequently employed in this discussion is applied to the disease of the mouth, especially in relation to the gums. Nothing is said in regard to other symptoms of the body, which would be necessary in order to make a clear diagnosis of scurvy. The logical inference is that such were not present.

Polar expeditions led to the conclusion that a diet of fresh or even raw meat, without any food or vegetables whatever, and associated with hardship, dirt and misery, or one consisting entirely of tinned, preserved and sterilized foods of the highest quality but with no fresh food, animal or vegetable, did not produce symptoms of scurvy, while scurvy appeared when, along with potatoes, etc., and daily doses of lime juice, the bulk of the food consisted of ordinary salt beef or pork. Until the recent Antarctic expedition, from that of Nordenskjold in the Vega, none had been attacked by scurvy except that of Jackson, whose men remained on board the ship where they had lime juice, potatoes, etc., but refused the coarse, even "gamey" bear's flesh, on which alone the exploring party subsisted. All were attacked with scurvy, two, indeed, dying. The Laplanders of Finland bartered for farinacea, etc., but ate their fish putrid by preference, and suffered much from scurvy.

In scurvy there is inflammation and bleeding gums; the gums puff up, thicken and bleed easily; the teeth become loose and sore upon mastication; a disagreeable odor comes from the

mouth; salivation or ptyalism results from irritation of the teeth, as well as scorbutic anaemia; the patient is languid or tires, perspires freely upon exertion, has shortness of breath and palpitation of the heart; the face is ashy gray, becoming paler each day; hemorrhage takes place in different parts of the body, especially beneath the skin, in the muscles and beneath the periosteum, as well as in the joints. This often gives considerable pain and sometimes causes inflammation, with resultant pus infection. Occasionally hemorrhage takes place in the internal structures. The temperature varies and both febrile apyretic states occur.

On the other hand, the symptoms observed in interstitial gingivitis are confined to the gums and alveolar process. There are no constitutional symptoms. This disease was formerly known as pyorrhœa alveolaris because the disease was not recognized until pus was observed about the teeth. It frequently exists for years before pus is noticed. All the teeth may be lost without pus.

From what has been said, it would seem rather difficult to assume that these mouth conditions herein described were entirely due to scurvy. Changes in climate and environment must be considered in relation to the interstitial gingivitis as well as unhygienic conditions.

It is common to find scurvy in private and state institutions where many people are confined, due to a monotony of food. The following scorbutic case was referred to me by Dr. George W. Johnson: A twenty-five-year-old American was admitted to Cook County Hospital for the Insane December 2, 1892, suffering with melancholia, attended by delusions of persecution and suicidal tendencies marked by refusal of food. June 1, 1896, he again began to refuse food, but took liquid diet on persuasion. June 29, the patient was transferred to the hospital because of his emaciation and scorbutic symptoms were discovered. July 18, the constitutional and local symptoms of scurvy were well marked. The teeth were covered with sordes and loosened. Under antiscorbutic treatment these symptoms had fully disappeared by August 13. Through the kindness of Dr. Johnson I was allowed to see this patient. I found none of the teeth very

loose, showing the disease was superficial. I removed two teeth that were decayed and the most loose. These were prepared for the microscope in the usual way. The gums and peridental membrane were in an active state of inflammation. Small blood vessels were observed in different localities with round cell infiltration extending into the tissue. The root of the right superior second bicuspid, with peridental membrane attached, showed active inflammation about an artery which had thickened, and an area of tissue degeneration, forming an abscess. The interstitial gingivitis due to scurvy, drug or self-poisoning has the same pathology.

CHAPTER XXI.

TOXINS PRODUCING TROPHIC CHANGES.

The retrograde disturbances of nutrition lead to degeneration of the affected tissue. Tissue infiltrations are due to deposits in the tissues of pathologic substances which have either been formed within the body or introduced into it from without. These disturbances of nutrition may affect the alveolar process during its period of development and growth, but more particularly in a fully developed state.

Poisons may be divided according to their action into three groups: First, those producing local tissue changes; second, those acting injuriously upon the blood; third, those affecting chiefly the nervous system and the heart without producing recognizable anatomic lesions. The poisons of all these groups, acting on the alveolar process, either by producing irritation of the blood vessels or by disturbing nutrition because they are stored in the tissue by producing trophic and vasomotor changes through their action on the nerves, work toward the ultimate end of its destruction.

All causes which bring about stasis of blood in the capillaries, such as inflammation, pressure, hemorrhage or blocking the venous outflow from the part, will cause arrest of nutrition. Again, if the arterial supply be cut off from any cause, destruction of the process will take place. Arrest of circulation need not be permanent. It suffices for its evil effect if it persists for a certain time. The more highly specialized a tissue, the briefer its vitality when deprived of blood; thus, when the blood supply is cut off, absorption of the alveolar process takes place before the circulation can be re-established. This is more than likely to take place in the alveolar process since it is a doubly transitory structure and an end organ.

Under the influence of poisons of all kinds, the alveolar process is liable to be absorbed. The result depends on the condition of the patient and the severity of the poison. The lowering

of vital resistance is proportional to the depth of the poisoning, and the weakened condition of the tissue invites microbic infection and multiplication, affording a suitable soil for invading micro-organisms which lead to the development of pyorrhœa alveolaris. This is true of the poisons entering the arteries of the process which cause an increased pressure of blood.

The toxins which leave an indelible stamp upon the alveolar process are divisible into those belonging to the condiments, foods, beverages, drugs, and those arising from occupation. Tobacco, alcohol, tea, coffee, opium, cocoa, cocaine, as well as mercury, lead, brass, potassium iodid, phosphorus, sodium chloride and other metals.

With tobacco, as with alcohol and opium, the statistic method generally proves fallacious when applied to degenerative effects. The most careful researches show that the typical effects occur as a rule after long continued use of tobacco, sometimes not until twenty years or more. While many smokers reach old age, many fail to live to old age because they are smokers. The skin is subject to itching and reddening; the nerves of taste are blunted and patches develop in the throat; loss of appetite, epigastric fulness, pain, vomiting and disturbance of bowel function are common. Menstrual disturbance occurs in women, and in female cigar-makers abortion and pluriparity are frequent. The sexual appetite is impaired, and sometimes sterility and impotence occur. Disturbed heart action, palpitation, rapid and intermittent pulse, precordial anxiety, weakness, faintness and collapse, with sclerosis of the coronary arteries of the heart and left ventricular hypertrophy occur often. Cigars and cigarettes produce irritation of the nose and mucous membrane, diminished smell, chronic hyperemia of the epiglottis and larynx, and sometimes of the trachea and bronchi, predisposing to tuberculous infection. Nicotine amblyopia is common, with central disturbances of the field of vision and slight color blindness. Often there is disorder of the ear tubes and congestion of the drum, with loss of auditory power and consequent noises in the ear. The central nervous system is affected. In high schools non-smokers progress faster than smokers. Child smokers, from nine to fifteen years of age, exhibit less intelligence and more

laziness or other degenerative tendencies. Adults have head pressure, sleepiness or drowsy stupor, depression, apathy and dizziness. There may also be ataxic symptoms, paretic weakness of bowels and bladder, trembling and spasms. Tobacco insanities, though comparatively rare in smokers, are common in snuffers and still more in chewers.[1] In the precursory stage, which lasts about three months, there are general uneasiness, restlessness, anxiety, sleeplessness, and mental depression, often of a religious type. After this occurs precordial anxiety, and finally the psychosis proper, consisting of three stages: 1. Hallucinations of all the senses, suicidal tendencies, depression, attacks of fright, with tendency to violence and insomnia. 2. Exhilaration, slight emotional exaltation, with agreeable hallucinations after from two to four weeks' relaxation, again followed by excitement. 3. The intervals between exaltation and depression diminish, and the patient becomes irritable, but otherwise not alive to his surroundings. Perception and attention are lessened. The patient may be cured in five or six months if he stops tobacco during the first stage. In a year or so he may recover during the second stage. After the third stage he is frequently incurable. As the patient becomes (especially by the use of the cigarette) an habitue before puberty, the proper development and balance of the sexual and intellectual system is checked. These patients break down mentally and physically between fourteen and twenty-five. The moral delinquencies, other than sexual, are often an especial tendency to forgery and deceit of parents. Frequently the insanity of puberty (hebephrenia) is precipitated by tobacco. The cigarette, if used moderately, may be a sedative, but, as used, is a stimulant, and is often made of spoiled tobacco, resembling in reaction morphine, and acting on animals in a somewhat similar manner. As tobacco turns the salivary glands into excretory glands, it leads to imperfect digestion of starch and to consequent irregular fermentation in the bowels, thus at once furnishing a culture medium for microbes, from which to form violent toxins, and likewise creating leucomaines, to damage a nervous system overstimulated by

[1] Annual of the Universal Medical Sciences, 1895.

nicotine. This is one great reason why those who use snuff and chew tobacco become insane more frequently than smokers, albeit these last are not exempt.

Statistics from the female employes of the Spanish, French, Cuban and American tobacco factories, while defective and somewhat vitiated by the coexistence of other conditions producing degeneracy, support the opinion that the maternal tobacco habit (whether intentional or the result of an atmosphere consequent on occupation) is the cause of frequent miscarriage, of high infantile mortality, of defective children, and of infantile convulsions. Tobacco, therefore, in its influence on the paternal and maternal organism, exhausts the nervous system so as to produce an acquired transmissible neurosis.

Alcohol has been repeatedly charged with being the greatest factor in degeneracy. The influence of alcohol on the individual must first be studied to determine its potency and method of action as a cause of race deterioration. Careful medical researches have shown that alcohol produces a nervous state closely resembling that induced by the contagions and infections, and often accompanied by mental disturbance. The acute nervous state to which the term "alcoholism" was applied by Magnus Huss has all the essential characteristics of the nervous state due to the contagions and infections or mental exhaustion. The action of alcohol may be limited to the central nervous system and thus produce hereditary loss of power. It may cause changes or degeneracies in the peripheral nerves which in the offspring find expression in spinal cord and brain disorder through extension of the morbid process. But for its deteriorating effects on the ovaries and testicles alcohol would be a most serious social danger. Through these, however, it tends to prevent the survival of the unfit rather than to develop degeneracy.

Professional tea-tasters have long been known to suffer from nervous symptoms. Very early in the practice of their occupation the head-pressure symptoms of neurasthenia appear. Tremor also occurs early. While changes in the optic nerve have not been demonstrated beyond a doubt, still eye disorders have been observed in the pauper tea-drinkers of the United

States and the tea-tasters of Russia, indicating similar changes to those produced by tobacco and alcohol. The tea-cigarette habit has these effects. Bullard[2] finds that tea has a cumulative effect. In his experience, toxic effects are not produced by less than five cups daily. The symptoms manifested are those of nervous excitement resembling hysteria, at times almost amounting to fury; nervous dyspepsia; rapid irregular heart action; heart neuralgia; helmet-like sensation and tenderness along the spine. James Wood[3] of Brooklyn found that ten per cent of those under treatment at the city hospitals exhibited similar symptoms. Of these sixty-nine per cent were females, and every symptom ascribed by Bullard to tea was seen by Wood in his cases, who also found that the women manifested irregularities in menstruation of a neurasthenic or hysterical type. He found that these symptoms were produced by one-half of the quantity of tea charged with these effects by Bullard. The Lancet, several years ago, from an editorial analysis of the effects of tea-tippling, took the position that in no small degree nervous symptoms occurring in children during infancy were due to the practice of the mothers, both of the working and society class, indulging in the excessive use of tea, the excess being judged by its effects on the individual and not by the amount taken. Convulsions and resultant infantile paralysis were frequently noticed among the children of these tea-tipplers. Observations among the factory population and the workers in the clothing sweat-shops show that tea neurasthenia, presenting all the ordinary symptoms of nervous exhaustion, is especially common. It is evident that tea produces a grave form of neurasthenia readily transmissible to descendants. In addition to its effects directly upon the nervous system, tea tends to check both stomach and bowel digestion, and this increases the self-poisoning which is so prominent a cause, consequence and aggravation of these nervous conditions.

Coffee exerts an action very similar to that of tea, although the nervous symptoms produced by it are usually secondary to the disturbances of the stomach and bowel digestion. Coffee

[2] Annual of the Universal Medical Sciences, 1889.
[3] Ibid.

produces tremor, especially of the hands, insomnia, nervous dyspepsia and helmet sensations. With the exception of certain districts of the United States coffee abuse is not carried to such an extent as tea, albeit in these, as in some portions of Germany, the habit is an excessive one. The conditions described result in Germany as frequently as they do in the United States. Mendel [4] finds that in Germany coffee inebriety is increasing and supplanting alcohol. Profound depression, with sleeplessness and frequent cortex headache, are early symptoms. Strong coffee will remove these temporarily, but it soon loses its effect and they recur. The heart's action is rapid and irregular, and nervous dyspepsia is frequent. L. Bremer of St. Louis has observed similar conditions among both Germans and Americans there.

Opium seems to be the Charybdis on which the human bark strikes when escaped from the Scylla of alcohol. Its abuse as a narcotic is much older than is generally suspected even among the English-speaking races. Murrell over ten years ago demonstrated that the inhabitants of the Lincolnshire fens had long employed opium as a prophylactic against malaria. The ratio of insanity in these regions proved to be very great. The same conditions obtained in central malarial regions of New Jersey and Pennsylvania, where the use of strong infusions of the poppy was common. The statistics of Rush [5] as to opium-caused insanity in Pennsylvania indicate that the percentage of American opium abuses at the beginning of the nineteenth century was very great. The drug differs in two important aspects from alcohol—it is nearer in chemical composition to nerve tissue, and the tendency to its use may be transmitted by the mother directly to the fetus, since it passes through the placenta very often unaltered. Opium is a more dangerous factor of degeneracy than alcohol, since the opium habitue must be in a continuous state of intoxication to carry on his usual avocation, while abstinence from alcohol is perfectly compatible with proper work on the part of the alcoholist. The opium habit is increased by the propaganda carried on by the habitues, who justify their position by urging the use of opium for any ailment, however

[4] Neurologisches Centralblatt, 1887.
[5] Observations on the Brain and Mind. Page 10, 1798.

trifling. Opium, like alcohol, causes nervous exhaustion similar to but greater than that of the contagions and infections. From the affinity of opium to nerve tissue; from its tendency to stimulate the heart, thus causing increased blood supply to the brain; from its action on the bowels and the increased resultant work of the liver, this nervous state is much intensified. Opium does not interfere with the structure and fecundation of the ovary and testicles like alcohol, hence the danger of the opium habitue's children surviving. Opium, when smoked, stimulates the reproductive apparatus and thus greatly increases the number of degenerates due to this habit, although the defects due to the inheritance of the habit and their consequences lessen survival.

While coca took its place but recently among the toxic causes of degeneracy, it was a factor of Peruvian degeneration long ere the discovery of America. Forty-three years ago [6] Europeans or people of European origin in different parts of Peru had fallen into the coca abuse. A confirmed chewer of coca, called a coquero, becomes more thoroughly a slave to the leaf than the inveterate drunkard is to alcohol. Sometimes the coquero is overtaken by an irresistible craving and betakes himself for days together to the woods and there indulges unrestrainedly in coca. Young men of the best families of Peru are considered incurable when addicted to this extreme degree, and they abandon white society and live in the woods or in Indian villages. In Peru the term "white coquero" is used in the same sense as irreclaimable drunkard. The inveterate coquero has an unsteady gait, yellow skin, quivering lips, hesitant speech and general apathy. The drug has assumed an unusual prominence in the field of degeneracy since the discovery of its alkaloid, cocain.

In both Europe and the English-speaking countries the world over a habit has resulted which, while much overestimated, is undoubtedly growing and aggravating as well as producing degeneracy. Many of the cases reported as due to cocain are, however, chargeable to the craving of the hysteric or neurasthenic to secure a new sensation, or the desire on the part of the opium or

[6] Johnson, Chemistry of Common Life, Vol. II.

whisky fiend to try a dodge of forgiveness by friends. The habit is very frequently induced by patent medicines taken to cure catarrh by the neurasthenic or to cure nervousness by the hysteric as well. As deformities of the nose passages predispose to "catarrh," patent medicines for local application containing cocain are frequently employed in the treatment of this supposed constitutional disease, with the result of aggravating the original degeneracy. The youth under stress of puberty frequently ascribes all his ills to catarrh, and for it often employs snuffs containing cocain, and his nervous condition is much aggravated thereby. Among the nostrums urged in the newspapers and magazines for this condition so often resultant from nerve stress alone is a snuff containing three per cent of cocain. From the description given by Johnson of the coquero there can be no doubt that tramps, wandering lunatics and paupers result from this habit to give birth to degenerates in the next generation.

It is a widespread opinion among dentists that in toxic cases the gums are the first tissues involved. The fact is, however, that when the salts of mercury are taken into the system, as noted elsewhere, they act directly upon the central nervous system; later occur nausea and vomiting, tremor in the arms and hands. Besides local nerve inflammation (neuritis), mercurial and brass poisoning produce paralysis agitans, and lead poisoning, drop wrist, etc.

Excessive secretions of the glands of the body, especially the salivary glands, later occur with rise in temperature, gingivitis with periosteal and peridental membrane swelling, thickening of the gums and loss of teeth. The central nerve system disturbance affects all other structures. Inflammation of the mucous membrane of the mouth, as well as of the gums, and of the alimentary canal, frequently occurs with sloughing of tissue. The kidneys become involved, and are unable to carry off the effete matter.

The cachexia, which resembles that of scurvy, is characterized by great debility, anemia, emaciation, alopecia, atrophy and coarseness of the nails, with pain in the muscles and joints.

Mercury is eliminated by all excretory organs for which it has a great affinity. The soluble salts pass out by the bowels. So

long as the excretory organs of the body eliminate mercury, the tissues are not affected. Small doses are eliminated, but continuation of dosage soon involves the nervous system, and afterwards the tissues of the body, especially the jaws. The first effect of mercury upon dogs is to produce vivacity and animation. This lasts for two or three days, when the limbs begin to tremble. The kidneys and bowels act at first freely. At the end of seven or eight days paralysis agitans occurs. There is constant trembling, whether awake or asleep; loss of appetite, with slight rise of temperature. At the end of two weeks the gums become inflamed at the margins. If the drug be continued, death occurs in about three weeks. The loss of flesh is remarkable. Miners working in mercury mines, and looking-glass makers, are all affected to a greater or less extent. The nervous system is always involved. The kidneys become diseased. The hair drops out. The miners think it a happy issue from their trouble when they have lost all their teeth, or even the molars. They are henceforth exempt from suffering so far as the teeth are concerned. Many are toothless at thirty-five.

Mercury taken by the mouth is found in the urine in two hours, and in the saliva in four hours. It appears in the urine fourteen hours after it has been applied to the skin.[7] Although it is believed to have passed entirely out of the system, it has been found in the brain, liver, kidneys and muscles. It is claimed that, like lead, it forms combinations with albuminoids in the tissues, for a time remaining inert, to be subsequently oxidized and returned to the circulation as an active poison. While a single dose of mercury may be rapidly eliminated from the system, repeated small doses distributed over a long period are not so eliminated on account of the thickness and occlusion of the walls of the capillaries, producing endarteritis obliterans, hence more or less of it is deposited in the tissue.

Lead enters the system through the alimentary canal, skin and respiratory tract. A longer time is required to produce plumbism (lead poisoning) than mercurial poisoning. Lead is stored up in the system in minutest quantities for an indefinite

---

[7] Twentieth Century Practice of Medicine, Vol. III, page 935.

length of time. Its effects are not manifest until the central and peripheral nervous systems have become involved, as evinced by the effect of plumbism upon the wrists. Occasionally, the chief seat of deposit is the liver or muscles. It is chiefly eliminated through the kidneys, and very slightly through the liver and salivary glands. Not until a considerable length of time has elapsed is lead traceable upon the gums. This usually occurs about the lower incisors and cuspids. This deposit (lead sulphid) is always in the tissue outside of the blood vessels. Plumbism causes trembling, nausea and vomiting. The patient loses flesh, becomes anaemic, and has great resultant debility.

The lead circulating in the capillaries accumulates, owing to impeded circulation resultant on the thickening of the coats of the vessel, producing occlusion. A bluish line upon the gums indicates that the system is completely saturated. Like mercury, lead collects in the mucous membrane upon the inside of the mouth, producing blue patches from a line to one-half an inch in length. Lead not only produces local irritation, but affects the peripheral nerves as well, producing atrophic changes; upon the capillaries a thickening of the inner coat results in endarteritis obliterans.

Lead produces in those exposed to the fumes a systemic nervous exhaustion, characterized by local paralysis about the wrist, as well as the general symptoms of profound systemic nerve tire. This may result, as was pointed out nearly half a century ago,[8] in acute insanity of the confusional type followed very often by mental disorder of a chronic type resembling paretic dementia. In some cases the patient recovers from the acute insanity to suffer thereafter from epilepsy. In other cases [9] an irritable suspicional condition also results, in which the patient may live for years, marry and leave offspring. This last condition and the epileptic are the most dangerous as to the production of degeneracy. The women employed in the pottery factories in Germany suffer, according to Rennert,[10] from a form of lead-poisoning which produces decidedly degenerative effects

---

[8] Tanquerel des Planches, Lead Diseases, American Edition, 1848.

[9] Kiernan, Journal of Nervous and Mental Diseases, 1881.

[10] American Journal of Obstetrics, Oct., 1882.

upon the offspring. These women have frequent abortions, often produce deaf-mutes and very frequently macrocephalic children.

Brass-workers suffer from a nervous condition very similar to that produced by lead. Hogden [11] of Birmingham and Moyer [12] have called attention to the grave forms of nervous exhaustion produced among brass-workers. The period during which the patient is able to pursue the occupation without breaking down is longer than that of the lead-workers. Women, like men, are exposed to this condition. The chief effects produced, so far as the offspring have been observed, are frequent abortions and infantile paralysis. The green gum is an early symptom.

Potassium iodide exerts a like toxic influence to lead and mercury, as its pathology is similar thereto, but it is of infrequent occurrence.

The employment of women in match factories and tenement-house sweat-shops is growing. The chief toxic effect of phosphorus is not the localized jaw necrosis. This is but an evidence of the progressive system saturation with phosphorus, and bears the same relation to the more dangerous effects of phosphorus that the "blue gum" does to the systemic effect of lead.

In adults, excess of sodium chloride in the blood from consumption of salt meats and fish has been noted with scurvy. For this reason Rawls, of Cincinnati, Ohio, believed that an excess of salt in the system produced gingivitis. Languor, depression, anaemia, with a rise of temperature, and enlarged joints with soreness are the first symptoms.

The effects of this disease upon the system are almost identical with those of mercury and lead. Bruise-like (purpuric) eruptions occur upon the skin and mucous membrane, on the serous membrane (notably the pleura, pericardium, meninges and synovial linings of the joints), mucous membrane of the mouth, stomach, intestines and bronchi.

Owing to the anaemia, vascular weakness and altered composition of the blood, edema is common both in the lungs and in the submucous and subcutaneous tissue, especially the feet and legs. The gums begin to swell with redness and fibrous thickening of

[11] Birmingham Medical Review, Jan., 1887.
[12] Medicine, May, 1904.

the deep layer, which cause protrusion, especially in the cases of degenerates. The blood vessels, especially the capillaries, become thickened, in some cases they are occluded, or erosion and ulceration occur. The patient becomes decidedly pale and markedly debilitated. The skin is dry and blanched. General emaciation is evident.

The mucous membrane and gums become swollen and bleed, stomatitis ulcerans results in greater or lesser degree. The tongue is at first swollen, then it becomes dry and hard. The gums are at first red and swollen. They bleed easily upon the slightest touch. Later they become pale and are irregularly larger, somewhat fungoid and friable, protruding between the teeth. They are quite tender to the touch. Ulcers appear on the buccal surfaces. The stomach becomes irritable, nausea and vomiting are common. Constipation occurs early and diarrhœa later appears.

CHAPTER XXII.

AUTOINTOXICATION IN INTERSTITIAL GINGIVITIS.

Every day we hear or read of individuals, in the prime of life and to all intents in good health, dying suddenly. Post-mortem reveals nothing whereby we may satisfy our minds as to the actual cause of death and the case passes into medical history as an obscure condition. If we were familiar with autotoxic states, I am sure many of these seemingly obscure conditions could be accounted for. Autointoxication is the rock upon which the human bark is wrecked and of which physicians know so little.

The human organism even in its normal state is prone to its own destruction by poisons. These poisons are formed within the organism itself or are taken into it in foods, liquids and drugs.

Selmi, the Italian toxicologist, gave the name of ptomaines to basic substances formed in putrefying animal matter. From their similarity to vegetable alkaloids, the ptomaines are often spoken of as putrefactive or animal alkaloids. Leucomaines are animal alkaloids formed by the metabolic processes of the organism.

The processes of intestinal putrefaction and the formation of physiologic and pathologic alkaloids afford an explanation of the pathogenesis of many diseases, the origin of which was obscure until recent investigations gave us the key by which their true nature may be understood.

We must admit the great impetus given to disintegrating processes in organic matter by bacteria. In no part of the body is this more true than in the alimentary canal. During the process of digestion, changes of a chemic, putrefactive and fermenting nature take place in the small intestine, which give the opportunity for the formation of poisonous substances, and these, when absorbed have an injurious effect on the system. There is some protection against this, however, if the liver, kidneys,

lungs and skin (the great sewers of the body) be not too deeply involved. When these poisons are not eliminated, or when any one of the eliminating organs becomes diseased and other organs are obliged to perform that function, these poisons are carried in the blood stream to the remote parts of the body. The organs most frequently involved as recognized by physicians, are the liver, kidney, heart, brain and eye, but the bony alveolar process, a doubly transitory structure and end organ and the dental pulp, which is the most perfect end organ in the body, are the first to record symptoms of disease.

If the disease involves the entire system and all the eliminating organs, the skin, kidneys, bowels and lungs are performing, or can be made to perform, their natural functions, many of the poisons are soon removed. On the other hand, if any one of these organs be involved, the process of elimination is slower, as the eliminating organs, which are not involved, must do all the work. At best this is imperfectly performed. A great part of these poisons is eliminated by the stools. Owing to the slow movements of the intestinal contents, much of the poison is absorbed by the mucous membrane. In faulty metabolism or tissue changes, toxins are produced which are absorbed and pass into the lymph and blood vessels. All poisons, producing intoxication, whether due to disease, tissue change, fermentation or infection, are of interest in their relation to interstitial gingivitis. Many of these auto-infections are of short duration and their intensity is not lasting. On the other hand, the toxic action of mercury, lead, brass, and of the products of syphilis, tuberculosis, scurvy, etc., is familiar to all. These poisons are of vital importance to the patient and physician, since they act quickly, but, from the viewpoint of the stomatologist, autointoxications of slow progress are the ones of vital importance. These substances are taken directly into the blood vessels and carried throughout the system. This has been repeatedly proven by Bouchard.[1]

That man is born free from microbes, was first demonstrated by Metchnikoff. Soon after birth the skin and mucous membrane

---

[1] Autointoxication in Disease.

become infected, either from the air or water used in bathing or both. On an examination of the intestinal contents an hour after birth, during warm weather, bacteria were found. Usually bacteria are not described until from twelve to twenty hours after birth. Micrococci and bacilli flourish independently of food, for they are found in the alimentary canal before nourishment has been taken. These microbes change in character when mother's milk or other foods are given the child. The bacillus bifidus appears with mother's milk. The colon bacillus, streptococci, staphylococci, lactic acid bacillus, etc., with cow's milk. Later, with the changes in diet, whether purely vegetable or animal, microbic flora grow rapidly in the intestinal tract. Vignas and Suckdorf have shown that an adult man passes from 30,000,000,000 to 50,000,000,000 of bacteria daily in the faeces. Many, perhaps most of these bacteria, are harmless in healthy individuals and the majority are dead. They become exceedingly virulent after accidents or injuries, such as gun shot, knife or other wounds, strangulated hernia and catarrhal conditions of the mucous membrane. Man therefore, is in constant danger of being infected. The injury resulting from these micro-organisms, is not from the bacteria themselves but from their toxins, the products which are absorbed.

Autointoxication, without other pathologic states, is due to the absorption from the gastro-intestinal tract, of toxic material. Absorption is favored by constipation and the toxic action is enhanced by hepatic insufficiency.

Before considering further the putrefactive changes within the intestinal canal, I wish to speak here of a subject about which I find little of note by previous writers, namely, changes in the digestive tract, due to evolution. Evolution is based upon the law of economy of growth, laid down by Aristotle, or use and disuse of structures. It is applicable here as in other parts of the body. Man, as a whole, has undergone rapid changes and is still undergoing greater and greater specialization. In no structure of the human body are these changes so great, owing to disuse as in the digestive tract. When organs are exercised, like the arms of the blacksmith, the hands of the oarsman, the legs of the mail carrier, they become enlarged and strong. On

the other hand, when organs are not used, like the little muscles of the ear, the small ribs, the little toes, the blood does not flow to the parts in proportion and arrest of development results. I have repeatedly shown many times the arrest of the face, jaws and teeth in the evolution of man, due to disuse. Civilization, by its custom of preparing food and etiquette in eating, has caused rapid degeneration of the jaws and teeth, resulting in irregularities and decay. The mastication of food is a lost art with many people, the salivary glands are not excited, arrest takes place and saliva containing ptyaline is scanty. Foods cooked and swallowed without mastication are taken into the stomach with the preparation of first digestion. The gastro-intestinal juices are required to perform all the work. Changes in the liver, either as to size, quantity of bile secreted or disease cause hepatic insufficiency. The same is also true of the pancreas and gastro-intestinal juices. The size of the stomach, the length and deformity of the intestine and last, but not least, the condition of the nervous system and the power of the muscular coats of the intestines to expel the contents from the body are to be considered.

The evolution of the rectum and anus from the placental and oviparous mammals is interesting, but is too broad a subject to be considered at this time. This evolution, however, in its relation to malformations and muscular tonicity, owing to man's upright position in his phylogeny must not be lost sight of in the study of gastro-intestinal irregularity.

The sedentary life, due to modes of living, has brought about many of these changes. The digestive apparatus has not had time to readjust itself to the new environment. Micro-organisms and pus germs which have accumulated in the mouth are taken into the stomach and intestines with every swallow. These may produce injurious results. While the process of digestion converts the protein or albuminoid substances of the food into peptones and then into animo acids, the putrefactive bacteria further forms alkaloidal and other poisons which pass into the blood.

Direct demonstration of this fact has been shown by many investigators. Planer, after ligating the colon found H_2S in the

blood of the portal vein. Carter has found indican in animals, the subjects of intestinal derangements. Bouchard, as well as Planer, has observed alkaloids, not only in the tissues, but in the blood. Poisons formed, not only in the intestines, but also those existing in the tissues, are also observed in the urine.

That an increase in intestinal putrefaction will cause a large quantity of toxic material to pass through the blood into the urine has been demonstrated many times. Stadeler in 1848 found phenol in the urine. Bauman in 1877 found phenol in fecal matter. In 1826, Tiedeman and Gmelin discovered a red colored substance in the duodenum which proved to be indol. Braconnot later found in the urine, indican derived from indol. Prof. Metchnikoff of the Pasteur Institute thinks old age is chiefly caused by two poisons, phenol and indol which are generated in the intestines including such diseases as arterosclerosis, cirrhosis of the liver, and interstitial nephritis. He might also have mentioned heart lesions and interstitial gingivitis. In 1872, Jaffe injected indol under the skin and afterward found indican in the urine. Later experiments by Senator failed to find indol in the meconium or indican in the urine of newly born infants. It is an established fact today that the variation of indican in the urine is governed by the quantity of indol in the faeces. In other words, the amount of indican in the urine depends upon the activity of intestinal putrefaction. In cholera, typhoid fever, intestinal obstruction, Hassal, Gubler, Robin, Carter, Jaffe found large quantities of indican in the urine. Senator showed indican in the urine in constipation.

Nencki gave a dog two grains of indol by the mouth, and in twenty-four hours there appeared diarrhea. Twelve milligrams of a one per cent solution administered subcutaneously to frogs caused death. One and five-tenths to two grams of indol administered subcutaneously to a rabbit in twenty-four hours proved fatal. By similar experiments Salkowsky found phenol and cresol in the urine. Especially was this the case in diarrhea and in intestinal obstruction. There is no doubt that in future investigations other poisons will be found in the stools and urine that produce marked poisonous effect upon the system. Products of putrefaction formed in the intestines, found in the urine,

must of necessity circulate in the blood throughout the system. There is a natural fermentation going on all the time in the intestines. In young and middle aged people, when the excretory organs are performing their office in a healthy manner, the kidneys, bowels, skin and lungs remove the poisonous products from the body. When, however, putrefactive products are formed in excess, or the excretory organs have lost their tonicity, has the system other means of preventing the accumulation of poisons in the blood? Certainly, the liver is intended to perform that office. This has been proven by Schiff. The experiments by G. H. Roger with alcoholic extract of rotten meat show that when injected into the portal vein it is one half as toxic as when introduced into the circulation.

Bouchard has shown that blood drawn from the portal vein of a dog kills a rabbit in a dose from thirteen to sixteen cubic centimeters per kilogram; that blood removed from the liver requires twenty-three centimeters. He has also shown that the injection of the extract of 2.5 grams of decomposing meat is sufficient to kill a man.

Many other experiments have been made by scientists showing similar results. In a summary of the research work, it is safe to say that the liver is intended to give protection to the system by destroying poisons especially those derived from the intestines so that the general system does not receive an amount of these poisons above what the excretory organs are able to remove.

When all conditions work in harmony, that is, when animal and man, after years of normal environment, have adjusted themselves, disease is less likely to result. When a change of environment, such as food, climate and soil, takes place, the animal or man becomes more susceptible to disease. Thus fifty-five monkeys died of tuberculosis in the Lincoln Park Zoo, Chicago, in one year, due to change in food and temperature and to confinement. House dogs are more susceptible to disease than street dogs. The Indian of North America has been, and is, dying rapidly from change in environment and food. This is true of other primitive races throughout the world.

Scandinavians in American cities are very susceptible to disease. The same is true of the Negro.

Many people are still in the primitive stages as regards their digestive apparatus. They have inherited an atavistic tendency in their large, well-formed jaws, muscles and teeth. They masticate food and enjoy it like the carnivora, tearing and chewing meat from a bone. The digestive apparatus is perfect, the bowels, kidneys, skin and lungs do their work normally, and they are in perfect health.

Many in whom the digestive apparatus is weak have progressed along the line of evolution. These people live a sedentary life one or two generations in advance of the tiller of the soil. They are in the transitory stage, not yet adjusted to the new environment. A third class, born of neurotic parents, have inherited deformed internal organs the secretions and action of which are not in harmony with each other. They do not chew their food and digestion is impaired. Their nervous systems may be impaired from the first, or may become involved as a result of faulty digestion and assimilation.

Studying the three classes singly, it is found that the members of the first class are healthy, that they can eat and drink everything and at all times, day and night. They can eat eight or ten meals a day, like the King of Portugal, and enjoy them. They can drink large quanities of alcohol or beer each day without difficulty. They are rarely ill. When the senile stage begins, while there are no marked symptoms, the excretory organs fail to perform their work properly. Interstitial gingivitis sets in, the teeth loosen, arteriosclerosis, kidney breakdown, uremic poisoning result and at from fifty-five to sixty-five death takes place from Bright's disease, diabetes, heart failure or apoplexy, the result of excesses.

The second class easily produce acute gastro-intestinal fermentation, autointoxication, and are subject to sick headaches, acid stomachs, gases in both stomach and bowels and constipation. They suffer with headache, migraine and vertigo, and often with nervous symptoms. In these cases special foods will upset the entire system. Fruits, raw as well as cooked, set up fermentation in the small intestines and putrefaction results.

Coffee, chocolate, cocoa, beer, and the inhalation of tobacco smoke will disturb the digestion, produce cold extremities, sick headaches in a few hours and not infrequently skin eruption.

The third class are not only subject to all the symptoms of the first and second, but frequently surgical operations are necessary to establish healthy relations between the digestive organs.

Deformities of the jaws and teeth are not uncommon. Macaulay portrays a vivid picture of such a state in Charles V of Spain. Among other physical deformities, he says, "At length a complication of maladies completed the ruin of all his faculties. His stomach failed, nor was this strange, for in him the malformation of the jaws, characteristic of his family, was so serious that he could not masticate his food, and he was in the habit of swallowing ollas and sweetmeats in the state in which they were set before him. While suffering from indigestion, he was attacked by ague. Every third day his convulsive tremblings, his dejection, his fits of wandering, seemed to indicate the approach of dissolution."

Prof. Russell H. Chittenden[2] in his experiments in physiological economy in nutrition, has shown that excess of proteids means waste, "but of far greater importance is the unnecessary strain placed upon the body by this uncalled-for excess of food material which must be gotten rid of at the expense of energy that might better be conserved for more useful purposes."

He has conclusively shown that body equilibrium can be maintained on half the daily intake of food. The brain worker and the muscle worker can maintain health, strength and vigor on a smaller amount of nitrogenous material than is usually consumed; "that an excess of food is in the long run detrimental to health, weakening rather than strengthening the body and defeating the very objects aimed at." This applies to people who have obtained their growth and not to children.

Some neurotics and degenerates are very susceptible to autointoxication on account of unstable nervous systems. They either become easily constipated or toxic material accumulates

[2] Physiological Economy in Nutrition.

in the intestines and as a result the system becomes slowly poisoned. Convulsions occur in both children and adults. Most, if not all, insane patients are constipated. While it would not be safe to say that the insanity was due to constipation, yet all are greatly benefited, and some slight forms are cured, by keeping the bowels free from microbic infection.

Some of the best specialists claim the skin eliminates very little of the blood's poison. Under ordinary circumstances the skin excretes water, salts in small quantities, carbonic acid, and in some volatile fatty acids. As age advances and the eliminating organs lose their activity, the bowels and kidneys fail to eliminate all the decomposed material. When these organs become diseased, the skin and lungs assist in carrying off the poisons or their products. The skin especially is important in keeping the system in a healthy condition, free from poisons.

What the laity understand as "spring fever" is but the readjustment of the eliminating organs from winter to spring. People in the senile stages feel better in warm climates than in cold, hence the custom of moving to warm climates in winter.

During pregnancy, poisons are formed in the mother and fetus which circulate in the maternal and fetal blood. Upon the mother is thrown the burden of eliminating by the kidneys, liver, intestines, skin and lungs the bulk of the poison formed within the two organisms. When these poisons are retained autointoxication is produced which varies in degree from heightening the arterial tension, headache, gastric disturbance, and lassitude to convulsive seizures as in puerperal eclampsia. Interstitial gingivitis is always present to a more or less marked degree. The urine under these circumstances usually contains albumin. That errors of diet often induce puerperal eclampsia, there is no doubt. Pregnancy frequently advances normally until some such improper food as lobster, fish, pork, pie, strawberries, etc., is eaten ravenously, when, as the result of the entrance into the blood of imperfectly digested products or intestinal poisons, eclampsia follows. The presence of these toxins in the blood induces structural alterations in the renal epithelia and as a consequence renal *debris*, tube casts, are present in the urine along with albumin. If the patient lives the morbid changes are,

for the most part, temporary, for they disappear on cessation of the pregnancy. We are familiar with the dropsical legs of women seen near the end of pregnancy, but it occasionally happens that there is in addition to the autointoxication from intestines and kidneys, a hepatic toxemia, as well.

An abnormal degree of urinary acidity extending over a period of nine months accounts for many of the neuralgias, toothaches, destruction of teeth by erosion, decay of the teeth and wasting of the alveolar process, skin diseases, and many other lesions so common in pregnancy which cease to trouble after birth of the child. Mental strain due to overwork, grief, shock, etc., check the secretions, causing an abnormal degree of urinary acidity which eventually results in diabetes, Bright's disease and arterial degeneration. There are many other lesions traceable to or influenced by a high acidity of the system.

The liver becomes enlarged and tender, the patient slightly icteric, the stools pale, fluid appears in the abdominal cavity, and there are albumin and bile in the urine. It is not until the pregnancy has been brought to a natural or an artificial termination that the symptoms and physical signs disappear. In such a case the liver has failed to arrest and destroy the intestinal poisons as they pass through it and the result is that owing to their excess in the blood and inability on the part of the kidneys to eliminate them the patient is poisoned by the products within her own body.[3]

One accustomed to the odor of the skin and lungs and to an examination of the mouth in which there is interstitial gingivitis, can readily detect intestinal fermentation and kidney irregularity. Many times I have detected a tendency to kidney lesions or the lesion itself, in this manner, as was confirmed by subsequent urinalysis.

Autointoxication in disease is familiar to us all. That the blood is charged with effete matter or poison, due to autointoxication, is abundantly proven. Owing to a swollen mucosa or other obstruction, ordinary nose breathing furnishes an insufficient supply of oxygen. More air is necessary, hence the uncon-

[3] Autointoxication in Disease.

scious opening of the mouth. A larger volume of air by nose and mouth is therefore taken into the lungs. Most people at the senile stage do this, most noticeably, however, at night.

The poisonous products of the intestinal canal not expelled from the bowels are absorbed and carried by the portal system to the liver. If toxic material is sufficiently modified by the liver, it will be carried back and emptied into the bowel along with the bile. If, owing to some mechanical obstruction, as catarrhal swelling, gall stones or thickening of bile, the normal function of the liver should be interfered with, hepatic insufficiency results. Any derangement of the bile or liver cells which interferes with the proper function of rendering harmful substances innocuous would cause abnormal and poisonous products to be carried in the blood.

The great outlet for poisons which the liver fails to eliminate are the emunctories, chief of which are the kidneys. If there be too much work in this direction, the eliminating function is soon lost. The toxic material accumulates and results in renal inflammation and albuminuria. When this has taken place the blood becomes charged with poisons, the heart and arteries undergo degenerative changes with cardiac hypertrophy and arteriosclerosis together with the consequent cardio-vascular diseases, insufficient blood supply to various vital organs, nervous disorders, Bright's disease, diabetes, rheumatism, gout, uric acid diathesis, skin eruptions and asthma result. Before these diseases have become of sufficient importance to be observed by the physician, interstitial gingivitis has obtained full sway. In all the above mentioned diseases interstitial gingivitis is most pronounced.

The effects of autointoxication on the system are to reduce its vitality or to destroy the tonicity of the nervous system, the result of which, end organs of the body, the kidney, the brain, the eye, the dental pulp and the alveolar process first become diseased. This disease is brought about by overstrain upon the peripheral nerves, change in chemical structure of their blood cells, irritations upon the coats of the blood vessels and a general weakening of the part. The organ first involved depends

upon its anatomy, its structure, its function, its weakness, locality, power of recuperation, etc.

The poisons circulating in the blood, due to autointoxication, collect in the peripheral blood vessels of the alveolar process, change the character of the red blood cells and prevent nourishment from going to the parts.

A low vitality of the structure is produced. Irritation of the vessel walls is set up and absorption of the process results. People in advanced years in comparative good health (not ill), who are attending to their various affairs each day but who do not throw off the poisons of the body as readily as they did formerly, have an absorption of the alveolar process beginning at the gingival border and slowly extending toward the apical end of the root or roots of the teeth. The severity of this interstitial gingivitis will depend upon the vitality of the patient and the degree of poisons circulating in the blood. How much more severe, therefore, must be the absorption when disease of one or more organs of the body occurs, especially the eliminating organs.

It has been shown that poisons originating anywhere in the alimentary canal have been found in the urine, that poisons entering the system subcutaneously have been found in the urine. Some of these poisons are modified in form and intensity of action, while others remain in their original state. It has been a mooted question just how these poisons are excreted by organs in which they are not formed but a sensible conclusion must be that they enter the blood and are conveyed by it throughout the system and partly eliminated by the kidney.

In a logical understanding of autointoxication and its treatment an examination of the urine should be made in order to ascertain the extent of poisons circulating in the blood.

CHAPTER XXIII.

URINARY SIGNS OF AUTOINTOXICATION.

That changes in the character of the blood composition take place from week to week is known to every physician. These changes may be due to systemic derangement or to the character of the food, and are often so great as to affect the character of the tissues of the body, especially the alveolar process. We have at present no definite method of testing, from time to time, the chemical constituents of the blood. The best and only method of obtaining an approximate knowledge is by an examination of the urine. For many years I have made a special study of the constitutional condition.

The examination of the urine is the only means at hand of ascertaining the general condition of the system underlying interstitial gingivitis. Two factors are of considerable moment, namely, an excess or diminished urinary acidity and indican. I wish to report three hundred and ninety-four examinations. The patients are from twenty-seven to sixty-seven years of age. All had interstitial gingivitis in its most aggravated form with loose teeth in varying numbers. Thirty-two had lost teeth as a result of the disease. Fourteen had pyorrhœa alveolaris that could be observed by the naked eye. Twenty-four hours' urine was obtained. A part or all was sent to the Columbus Medical Laboratory for examination. In tabulating the reports, the following results were obtained: Specific gravity taken in the first fifty only, showed two had 1,005; two, 1,006; two, 1,008; two, 1,009; one, 1,010; one, 1,011; one, 1,012; two, 1,013; six, 1,014; one, 1,015; five, 1,016; two, 1,017; one, 1,018; five, 1,020; three, 1,023; one, 1,024; three, 1,025; one, 1,026; two, 1,027; one, 1,028; two, 1,029; one, 1,031. There were granular casts in six reports; hyaline casts in twelve; cylindroid in twenty-two.

The degree of acidity was obtained by taking 10 c.c. of urine specimen, measured in the graduate glass, then placed in the small glass; four drops of phenolphtalein were added; then drop by drop NaOH (1-10 normal sodium hydrate) until a slight pink-

ish color was produced. Having noted on paper the number of c.c. of the NaOH in the burette before and after the pink color was obtained, the number of c.c. displaced multiplied by 10 (in order to find the number of c.c. NaOH necessary to neutralize 100 c.c. urine) equaled the degree of acidity. Each step in this operation must be carefully performed, each instrument must be kept perfectly clean in order to get good results.

The results showed two had 4 degrees; two, 6; one, 7; two, 8; nine, 10; three, 11; seven, 12; one, 13; eighteen, 14; five, 15; twenty, 16; five, 17; two, 17.5; fifteen, 18; twenty-seven, 20; four, 21; thirteen, 22; six, 23; fourteen, 24; seven, 25; fifteen, 26; one, 27; seventeen, 28; one, 29; thirty, 30; two, 31; nineteen, 32; sixteen, 34; six, 35; twenty-seven, 36; five, 37; eleven, 38; two, 39; twenty-two, 40; two, 41; eight, 42; nine, 44; two, 45; ten, 46; two, 47; seven, 48; one, 49; eight, 50; one, 51; six, 52; two, 53; one, 54; one, 55; fifteen, 56; one, 57; five, 58; two, 59; seven, 60; six, 62; three, 63; four, 64; three, 66; two, 68; four, 70; three, 72; two, 74; five, 75; one, 76; one, 78; one, 79; one, 80; one, 83; one, 84; two, 100; three, 104; one, 105; one, 108; one, 110; one, 113; one, 120; three, alkaline; two, neutral. The urea showed two had .3 per cent; one, .5; two, .6; two, .7; two, .9; two, 1; one, 1.1; two, 1.3; one, 1.4; four, 1.5; six, 1.6; one, 1.7; three, 1.8; one, 1.9; one, 2; three, 2.1; three, 2.2; two, 2.4; six, 2.5; two, 2.6; one, 3; one, 3.1; one, 7.1. Albumin was found in four cases; blood in six; leucocytes in forty-five; epithelial cells in forty-six; uric acid crystals in two; urates in five; oxalates in fifteen. Of the three hundred and ninety-four examinations, three hundred and twenty showed indican to a greater or lesser extent. Seventy-four were normal in this respect.

To make a more complete study of each individual case, the following table has been prepared:

Specific Gravity.	Casts. Granular.	Hyaline.	Cylindroid.	Acid-Degree.	Urea-Per cent.
23	0	0	0	60	2.5
20	1	1	1	56	2.5
8	0	0	1	20	1.
6	0	1	1	12	.6
12	0	0	0	36	1.5
31	0	0	0	44	2.6
29	1	1	1	46	2.2

Specific Gravity.	Casts. Granular.	Hyaline.	Cylindroid.	Acid-Degree.	Urea-Per cent.
16	1	1	1	52	2.2
11	0	0	1	17.5	1.4
17	0	0	0	17.5	1.6
27	0	1	1	62	2.1
20	0	0	1	24	2.2
25	0	1	0	44	2.4
9	0	0	1	16	1.5
25	1	1	1	40	3.1
29	1	1	0	58	2.6
14	0	0	0	36	2.
16	0	0	0	30	1.8
14	0	0	0	30	1.5
13	0	0	0	36	1.6
13	0	0	0	36	1.6
15	0	0	0	36	1.7
15	0	0	1	15	1.6
14	0	0	0	36	.9
28	0	0	0	36	3.
5	0	0	0	degree not taken	.3
16	0	0	0	"	1.6
20	0	0	0	"	1.5
10	0	0	0	"	1.1
25	0	0	1	"	2.5
27	0	1	1	62	2.1
25	0	0	0	degree not taken	2.5
14	0	0	1	30	1.3
20	0	0	0	degree not taken	1.9
24	0	0	1	"	2.4
26	0	0	0	"	2.1
18	0	1	1	"	1.6
14	0	0	1	30	1.8
5	0	0	0	15	.7
23	0	0	1	40	1.8
7	0	0	0	14	1.3
16	0	0	0	30	1.8
9	0	0	1	11	.9
14	0	0	1	22	1.5
7	0	0	0	20	.7
7	0	0	0	20	7.1
23	0	0	0	60	2.5
20	1	1	1	56	2.5
8	0	0	0	20	1.0
6	0	1	1	12	.6

TOTAL EXAMINATION OF URINE.

Qualitative Examination.

Physical condition:

Clear 17

Cloudy 33

Present 30

Absent 29

Albumin:

Reaction:	
Acid	46
Alkaline	4
Color:	
Yellow	46
Amber	4
Odor:	
Negative	46
Present	4
Mucin:	
Present, trace	4
Absent	46
Peptones	None
Sugar	None
Bile	None
Blood:	
Present	6
Absent	44
Indican	50

Microscopical Examination.

Casts:	
Hyaline	14
Granular	7
Epithelial	1
Cylindroids	21
Cells:	
Blood	4
Leucocytes	45
Pus	11
Epithelial cells	46
Crystals:	
Uric acid	2
Urates	5
Oxalates	15
Phosphates:	
Calcium	1

The relation of acid autointoxication and mouth acidity is very intimate. The acids taken into the body and those produced by chemical changes within, such as hydrochloric, lactic, acetic, diacetic, oxybutyric, uric, and other acids circulate in the system in the form of salts so that the blood maintains at all times essentially a neutral reaction. If the acid ions in the blood at any time overbalance the metallic ions so that there is a considerable number of H ions present, an excretion of acid takes place and passes out through the kidneys, lungs, skin, and mucous membrane, especially of the mouth. If the kidneys do not carry off the surplus acidity, a greater strain is put upon the lungs, skin and mucous membranes of the mouth. The alveolar process and gums, being doubly transitory, as well as end organs, contain excretive and secretive glands. The gums are the first structure of the body which indicate systemic defects particularly noticeable in mercurial, lead, and brass poisoning, scurvy, etc. The mucous glands normally excrete acid fluid, while the salivary glands secrete alkaline fluid. It not rarely happens, however, that the kidneys fail in their function and the system becomes so saturated with acid that the salivary glands continue to cause destruction of the teeth.

Friction of the lips, teeth, and foreign bodies assist greatly in tooth destruction. Teeth softened by faulty nutrition and acid

states are easily destroyed by acids and friction.

In an examination of the urine of diabetics, tabetics and paretic dements, I have the following statistics:

1. *Urinalysis of Diabetic Patients.*—Urinalysis by the Columbian Medical Laboratories of three hundred and ninety-four diabetics showed specific gravity, one 1.003, one 1.005, one 1.007, one 1.010, one 1.011, eight 1.012, one 1.013, six 1.014, six 1.015, four 1.016, five 1.017, seven 1.018, six 1.019, seven 1.020, five 1.021, thirteen 1.022, fifteen 1.023, ten 1.024, twenty 1.025, thirteen 1.026, thirteen 1.027, eighteen 1.028, nineteen 1.029, twenty-five 1.030, twelve 1.031, fifteen 1.032, twenty-eight 1.033, sixteen 1.034, twenty 1.035, seventeen 1.036, eighteen 1.037, nine 1.038, twelve 1.039, ten 1.040, eight 1.041, eight 1.042, two 1.043, seven 1.044, two 1.045, two 1.046.

Percentage of Sugar.—Twelve had 0.1 per cent, eighteen 0.2, three 0.3, eleven 0.4, seven 0.5, seven 0.6, one 0.7, four 0.8, eight 0.9, ten 1, one 1.1, eight 1.2, seventeen 1.3, six 1.4, one 1.5, thirteen 1.6, seven 1.7, four 1.8, one 1.9, five 2, three 2.1, six 2.2, three 2.3, seven 2.4, three 2.5, five 2.6, one 2.7, three 2.8, three 2.9, four 3, one 3.1, five 3.2, six 3.3, one 3.4, two 3.5, ten 3.6, five 3.7, five 3.8, fifteen 4, four 4.1, five 4.2, two 4.3, two 4.4, three 4.5, eight 4.6, four 4.7, five 4.8, five 4.9, five 5, two 5.2, ten 5.3, five 5.4, one 5.5, nine 5.6, seven 5.7, five 5.8, one 5.9, nine 6, five 6.1, ten 6.2, ten 6.4, one 6.5, five 6.6, one 6.7, three 6.8, five 6.9, three 7, one 7.1, two 7.2, two 7.3, two 7.4, one 7.6, one 7.7, three 7.8, one 7.9, one 8, one 8.2, one 8.5, one 8.7, one 9, one 9.1.

Degree of Acidity.—Two passed 4 degrees, two 6, one 7, two 8, six 10, five 12, sixteen 14, one 15, fifteen 16, one 17, thirteen 18, twenty 20, one 21, twelve 22, one 23, ten 24, two 25, fourteen 26, sixteen 28, one 29, twenty-one 30, seventeen 32, thirteen 34, four 35, twenty 36, two 37, eleven 38, two 39, thirteen 40, one 41, eight 42, seven 44, two 45, eight 46, two 47, five 48, five 50, five 52, one 54, twelve 56, one 57, four 58, five 60, one 62, one 63, three 64, two 66, two 68, two 70, three 72, one 74, one 75, one 100, two 104, one 120, one alkaline, two neutral.

Acetone.—Of this number of cases only nineteen were examined for acetone. In eleven, acetone was present, in eight absent. Thirty-two were examined for diacetic acid; in six it was pres-

ent and in twenty-six absent. Twenty-four were examined for oxybutyric; in all it was negative.

2. *Urinalysis of Tabetic Patients.*—Degree of acidity in thirty-five was as follows: One passed 5 degrees, one 6, one 7, three 9, two 10, one 11, one 14, one 17, two 19, two 20, one 22, one 46, one 48, one 49, one 50, one 56, one 62, one 73, two 76, one 78, one 81, one 82, one 84, one 97, one 99, one 112, four alkaline. Those patients having the alkaline urine had marked erosion of the teeth showing that at some time there had been a high degree of acidity. Cystitis caused the urine to become alkaline. All showed indican to a greater or less extent.

3. *Urinalysis of Paretic Patients.*—Degree of acidity: There were twenty-one males, four females. Three passed 5 degrees, one 7, three 8, two 9, one 10, one 11, one 12, one 13, one 15, two 16, one 17, one 22, one 28, one 34, one 38, one 39, one 44, one 51, one 52, one 70. These patients were in a quiet state. If the urine could have been examined after excitement or an explosion the degree of acidity would have been greater.

4. *Urinalysis of Private Patients.*—Degree of acidity: I examined one hundred and twenty-nine. Three were also sent to me by Dr. J. F. Keefe of Chicago, making in all one hundred and thirty-two. They were from eleven to eighty-four years of age. All showed erosion and abrasion to a greater or less extent. Three passed 2 degrees, three 8, two 10, two 11, seven 12, two 14, two 15, five 16, six 18, two 19, eight 20, five 22, five 24, six 26, four 28, two 29, six 30, two 31, four 32, two 33, three 34, nine 36, two 38, two 40, two 44, five 46, one 47, two 48, two 50, five 52; five 54, four 56, one 58, three 60, two 62, five 70, one 90, one 127, one 132, only four or 3.8 per cent had uric acid. I quote here from a previous paper, ''Interstitial Gingivitis Due to Autointoxication,'' my first fifty patients' degree of acidity, one had 11 degrees, two 12, one 14, two 15, one 16, two 17.5, four 20, one 22, one 24, five 30, seven 36, two 40, two 44, one 46, two 56, one 58, one 59, one 60, two 62; 3 per cent had uric acid, all had indican.

The acidity of a single specimen of urine will vary like the specific gravity within large units, corresponding to the amounts of acid entering the blood from various sources. The normal degree of acidity of the urine is from 30 to 40 degrees. A low

acidity may arise from several factors: 1. A large excretion of water, as in nervous states, diabetes insipidus, etc. 2. A diet containing a large quantity of salts of the vegetable acids. 3. A corresponding deficiency of meat which yields acid salts. 4. A deficient power of the kidney to excrete acid. 5. Excessive elimination of acid by other emunctories. In patients in whom the degree of acidity exceeds 40°, there is excessively imperfect oxidation which, irrespective of the types of acid, underlies, as is now pretty generally recognized, severe constitutional stress allied to that of diabetic acidosis.

The quantity of urine passed in twenty-four hours influences the degree of acidity. Thus, if more than 40 ounces (about the normal amount) be passed, the degree of acidity with the same total amount of acid would be low as compared with less than 40 ounces.

On application of the phenolphtalein, if the urine specimen turns pink, it is alkaline, therefore no degree of acidity can be obtained. Litmus paper is applied to the gums and lips to ascertain if the mucus be acid. Acid mucus was found in every case tested. The circle of evidence, therefore, is complete.

Few adult persons have not had an excess of acidity at some period. Complete oxidation is essential to a normal condition. The organs and tissues of the body act as best they can to bring about this condition. In some systems, the liver has all it can do to care for the waste products of the tissues themselves. That the salts of fruit acids may be converted into alkaline substances in the system is true. The liver and tissues become overworked. The fruit habit (especially grape fruit) so generally indulged in to excess in America is producing havoc with the alveolar process, gums and teeth.

One case is sufficient illustration of the many requiring treatment. A twenty-seven-year-old woman had her teeth and mouth put in good condition in January, 1907. February 16 she returned with what she thought a cavity at the cervical margin of the left superior cuspid. Upon examination, I found the gums inflamed and receding, not only at that particular location but about all the teeth. Previous to this, the gums and mucous membrane were in fairly good condition. Litmus test showed the

mucus to be very acid. There was no cavity, only sensitive exposed dentine. Much gas was passing from the stomach. Upon interrogation in regard to her food she informed me she had been eating grape fruit every morning for three weeks. Urinalysis of a twenty-four-hour specimen showed the degree of acidity to be fourteen. Sixteen degrees were retained in the system. The recession of the gums and the sensitive dentine were due to the acid retention. The skin, lungs, and mucous membranes try to dispose of the surplus. If these structures are unable to do so, they are expelled as gas, vomit, or fermentative stools. That the acid excess of the system does pass through the mucous and salivary glands of the mouth to produce destruction of tissue, has already been demonstrated. With these illustrations of tissue in the mouth the question arises how far does this acidity affect other tissues and diseases of the body? For want of time, merely a few urinalyses in diseases were made.

Disease.	Degree of Urinary Acidity.
Arthritis (rheumatoid)	One 70.
Backache (severe)	One 10; one 25; two 30; one 35; one 36; one 40; one 50; one 79; one 82; one 87; one 90; one 95; two 100; two 110; one 120.
Bronchitis	One 56; one 58; one 60; one 61; one 67; one 120.
Constipation	One 25.
Coryza (acute), children 3 to 13 yrs.	One 12; two 13; one 14; one 15; one 16; one 17; one 20; one 22; one 26; one 27; one 30; one 36; one 46; one 47; one 50; one 56; three 58; one 70; one 72; one 90.
Cystitis (acute)	One 50.
Diphtheria	One 28.
Dipsomania	One 44.
Eczema (hands)	One 50; one 80.
Enlarged prostate	One 100.
Enterocolitis (chronic)	One 35; one 80; one 110; one 112.
Exophthalmic goiter (puberty stress)	One 54.
Fibrillae tremor	One 21.
Gastric hyperchloridice	One 60.
Gonorrhea	One 120.
Grippe	Two 25; one 40; one 50; one 60; one 75; one 76; one 80; one 82; one 88; one 90; one 94; one 97; one 110.
Hypertrophic rhinitis (acute)	One 21; one 25; one 113.

Hypertrophic rhinitis (chronic)	One 32; one 57; one 90.
Laryngitis (chronic)	One 65; one 85.
Middle ear, inflammation of	One 25; one 32; one 90.
Middle ear, chronic suppurative inflammation of	One 90.
Ménière's disease	One 65.
Migraine	One 40.
Myocarditis (chronic)	One 80; one 100; one 112.
Neuralgia following grip	One 20; one 24; one 25; one 27; one 40; one 46.
Pneumonia	One 18; one 26; one 27; one 33.
Pregnancy	One 10; one 12; one 14; one 16; one 18; one 29; one 30; one 50; one 57; one 60; one 62; one 64; one 67; one 82; one 84. one 82; one 84.
Rheumatism and gout	One 14; one 15; one 18; one 19; one 20; one 24; one 27; one 35; one 52; one 57; one 61; one 67; one 70.
Rheumatism and heart trouble, 8 years of age	One 40.
Scarlet Fever	One 33; one 80.
Sciatica	One 55; one 108; one 132.
Sphenoid sinus, inflammation of	One 57.
Sunstroke	One 20.
Tired feeling	One 25; one 40.
Tonsilitis (ulcerating)	One 10; one 12; one 14; one 20; one 27; one 48; one 72.
Tuberculosis	One 16; one 20; one 21; one 36; one 44; one 52; one 56; one 78; one 81; one 88; one 102; one 108; one 115; one 142.
Tuberculous hip disease	One 80.
Typhoid Fever	One 76; one 88; one 90; one 109; one 120.
Urticaria	One 47.

On comparing the office patients with those of other specialists and patients ill at home or in a hospital, it is found that the degree of acidity does not vary to any great extent. A constant abnormal degree of urinary acidity, in an individual attending to his affairs, means that sooner or later an organ or structure is bound to give way. This is particularly true at the senile period of stress (about sixty) when the arteries degenerate. The victim of an abnormal degree of acidity is more subject to disease than one with normal acidity. Study of the effects of a high degree of acidity in an otherwise normal individual whose teeth

and alveolar process are being destroyed has exceedingly interesting results.

Lessened blood alkalinity affects the whole alveolar process by setting up an irritation and inflammation of the coats of its arterioles and in the tooth pulp, producing endarteritis obliterans, arteriosclerosis, and nerve-end degeneration. I have demonstrated those diseases many times. Disease of the terminal nerves and arteries causes absorption of the bone. The inflammatory process has been termed interstitial gingivitis; the bone absorption, osteomalacia, or senile absorption, although it may occur early in life.

May not osteomalacia in other parts of the body be due to the same cause?

Cylindruria [1] is not necessarily associated with definite pathologic alterations of the renal parenchyma. This statement should likewise be accepted as to the occurrence of purely hyaline casts and their presence in small numbers. A few renal epithelial cells may be found at the same time occurring either in the urine or adhering to the casts, but never presenting an atrophic or otherwise altered appearance in the absence of definite renal lesions. The presence of compound hyaline and coarsely granular casts, as well as of waxy and amyloid casts, on the other hand, may be regarded as indicating definite changes in structure, so that as far as diagnosis is concerned microscopic examination of the urine furnishes information of more value than the simple demonstration of albumin.

Hyaline casts are more frequently seen—reference is here made only to the purely hyaline or, at least, but faintly granular form—and are found in all conditions in which albuminuria occurs. When present in only small numbers, and particularly when occurring but temporarily in the urine, it may be assumed, in the absence of other symptoms pointing to renal disease, that there is a mild circulatory disturbance of the kidneys.

The significance of blood and epithelial cells imbedded on hyaline casts is the same as the significance of blood and epithelial casts; both are pathologic and indicate nephritis.

---

[1] Clinical Diagnosis, p. 620.

Fine granular and hyaline casts often occur from auto-toxic strains on congenitally insufficient kidney in arthritic and allied states.

The presence of albumin in these cases was exceptional, it being found in but four cases. This would show that in none of these cases had the disease become very marked. When present it does not in itself indicate grave disorder, since albumin may be due to many conditions of the renal tract. It is of interest to us since disturbance of circulation may bring about albuminuria without inducing structural change in the kidneys. Purdy says: "Circulatory disturbances, in order to induce albuminuria, must include the renal vessels. In nature they must consist of acceleration of the arterial current or slowing of the venous current, in either case resulting in increased blood pressure. Again, in some derangements of the nervous system which interfere with the vasomotor nerve regulation of the renal vessels, temporary albuminuria is not an uncommon result." Albuminuria is present in auto-toxic neurasthenia, epilepsy, paretic dementia, and the renal crises of locomotor ataxia.

The specific gravity ranges from 1.005 to 1.031. The normal specific gravity ranges from 1.015 to 1.025. The difference depends upon the amount of solids and fluids present, increasing as the solids increase, decreasing as the amount of fluids increase. Specific gravity is hence an index in a general way of metabolic change. The low degree of acidity would indicate that a certain amount of acid was circulating throughout the system.

Indicanuria denotes the presence in the urine of potassium indoxyl sulphate formed by metabolism of indol absorbed from the intestines. It is supposed to be due to three sources: First, to intestinal putrefaction of nitrogenous substances; second, to suppuration in some part of the body; and third, to the formation of indol in the cells of the body-tissues. The fact that indican is found in the urine is a sufficient indication that this poison has circulated in the blood throughout the entire system and has been returned to the kidneys to be expelled. I have demonstrated many times that indicanuria and neurasthenia are in some way related. Reducing the intestinal putrefaction by the use of intestinal antiseptics, the neurasthenic condition of the

patient is often relieved. The toxic effect of acidosis and indican upon local tissues, especially upon terminal and transitory structure, is very marked. Their injurious effects consist of irritation of the coats of the blood-vessels and changing the chemical quality of the red blood cells. The salivary glands, the mucous glands, the alveolar process, and the dental pulp are the first structures to become involved.

Indicanuria is one of the great sources of autointoxication. The toxins of indican permeate all the structures of the body, being carried by the blood circulation. While acidosis and indicanuria may go hand in hand, the quantity of indican depends to a certain extent upon the acidosis relative to the amount formed in the intestines. Thus, upon reducing a high degree of urinary acidity to normal or below, the indican will be increased, owing to the abnormal bacterial activity in producing putrefaction. These germs seem to thrive better in alkaline than in acid media.

An accumulation of indican in the organism will often cause febrile disturbances, lassitude and gastrointestinal irritation—depending, of course, upon the severity of the attack. The effect of indican is not unlike that of poisonous drugs such as mercury, lead, phosphorus, bromine, quinine, etc., which produce poisonous symptoms in some individuals. In others there are apparently no ill effects. It must, therefore, not be overlooked that in all persons with persistent indicanuria the poison is continuously absorbed from the intestines into the circulation for months and years, and that in many persons it will not manifest itself until the periods of stress at forty-five and again at sixty years of age. Metchnikoff, in considering the phenomena of old age, concludes that autointoxication due to intestinal putrefaction is one of the most important causes of premature senility, in that it causes arteriosclerosis. The accumulation of these toxins in terminal organs, such as the pulp and alveolar process, is as disastrous as the accumulation of any of the poisonous drugs. Arteriosclerosis is one of the common diseases found in the alveolar process and in the pulp, which is a positive proof of its systemic origin.

To obtain the amount of indican in a given specimen, take

5 ccm. of urine; pour it into a test-tube; add 5 ccm. of hydrochloric acid, and shake thoroughly. Let the mixture stand for a few moments. Add 10 drops of hydrogen dioxide, shake thoroughly, and let it stand for a few moments; then add 1 ccm. of chloroform, shake thoroughly again, and let it stand. If indican be present, chloroform will absorb it, turn blue, and settle to the bottom of the tube; if there be no indican, the chloroform will remain colorless.

The toxins in the blood which accompany acidosis and indicanuria are carried to all structures of the body. All structures of the body, however, are not alike. Some, especially those that are active and are needed for the welfare of the body, under the law of economy of growth or use and disuse of structures, can take care of the toxins and return the blood to be cleansed of its impurities. Other structures which are of little use, and are called terminal organs and transitory structures, such as the dental pulp and the alveolar process, cannot dispose of the blood so readily. The result of this is an accumulation of toxins, and disease follows. Other terminal organs that can stand the strain a little better, but are sure to succumb later if the toxins continue to be present in the blood, are the kidneys, the heart, the liver, the eye and the brain.

I wish to call the attention of the profession to the fact that early symptoms and systemic disorders may be recognized, and prophylactic means may be adopted to ward off future trouble. Heart-pressure, endarteritis obliterans, arteriosclerosis and dilated arteries are easily demonstrable in the pulp and in the alveolar process by early symptoms of acidosis and indicanuria. It will be seen, then, that acidosis and indicanuria are factors which cannot be overlooked.

Critical examination of tables must convince a careful observer that in every examination two conditions are present; first, autointoxication due to intestinal fermentations and faulty elimination as represented by the indican and an abnormal urinary acidity degree; second, kidney over-strain and renal insufficiency due to hepatic insufficiency. When the liver fails to destroy the poisonous materials and the bowels to eliminate the toxins, over-strain of the kidneys causes the blood to become

overcharged with toxins and acidity, the heart and arteries undergo degenerative changes, and cardiac hypertrophy and cardio-vascular diseases, with insufficient blood supply, result.

It may be possible that in the near future other poisons than those already mentioned will be found in the urine, which may produce toxic effects on the alveolar process. Since this chapter was written, the following appears in the Journal of the American Medical Association: "A few unique anomalies of metabolism, notably alkaptonuria and cystinuria, have attracted attention quite as much because of the interpretation which they lend to the normal disintegration of protein in the body as on account of the pathologic features involved. From the fact that certain diamins, cadaverin and putrescin, characteristic of the putrefaction of proteins, frequently are found in conjunction with cystin in the urine of patients, it has at times been assumed that there is an essential connection in cystinuria between alimentary putrefactive changes and the output of characteristic abnormal excretory products. Cystinuria, however, has been regarded of late rather as an abnormality of protein metabolism in which the amino-acid cystin—a typical degradation product of albuminous substances—is not further broken down and oxidized as ordinarily. This view is strengthened by the simultaneous finding of other amino-acids, leucin and tyrosin, in certain cases. The latest novelty is the discovery of another amino-acid, lysin (diamino-caproic acid), in the urine of a patient with cystinuria. Since this compound is known to be the mother substance of cadaverin, the intimate inter-relation of the various observed constituents is further emphasized. Taken together, the accumulated data strengthen the conception of cystinuria as a condition in which the usual progress of protein catabolism is profoundly inhibited. One by one the unused fragments are cropping out as new cases become available for study."

CHAPTER XXIV.

ARTERIOSCLEROSIS, ENDARTERITIS OBLITERANS AND NERVE END DEGENERATION.

Endarteritis obliterans and hypertrophy of the middle and outer coats of the arteries are physiologic processes concerned in the disappearance of blood vessels functional in the fetal state, but losing such function after birth. Like all physiologic processes of the fetal type these become pathologic under ordinary conditions of post-natal life. For these reasons they again become physiologic in the involutional periods like the climacteric and senility. In transitory structures, like the alveolar process, there is continual trembling between the physiologic and pathologic. Undue excitation of the structure brings on an intensity of the process which tends to become pathologic. As I demonstrated a decade ago, endarteritis obliterans and hypertrophy of the middle and outer coats of the arteries play a large part in interstitial gingivitis.

In consideration of this subject, the changes in the elasticity of the artery and vein walls and the permeability of the capillary walls will be discussed.

The vessel walls are capable of great elasticity and bear the tension of the pressure of the blood without showing any condition of being strained. This elastic function of the walls is controlled by the muscular coat, which changes the chemic energy of the blood stream into energy of elasticity and in this manner keeps the vessel walls in a normal elastic state. The changes that take place in the arterial walls of the aortic system are of more or less importance, since the normal elasticity of the artery walls, blood pressure regulation, and distribution are dependent upon the depth and intensity of these changes.

When the vessel walls become diseased there may or may not be serious disturbance in the blood circulation. The acute and chronic contagions and infections, a general nutritional disturbance or poisons taken either internally or externally, and a high

blood pressure tend to weaken the arterial walls so that their elastic tonicity, in some instances, is completely lost.

This weakened elasticity is easily recognized by a distensile pulse and also registered by the sphygmomanometer. At the same time, arterial murmurs can be heard in the large arteries, especially the femoral, which are apparently a result of the rapid vascular changes characteristic of a high pulse. A pulse sometimes observed in retinal arteries can also be attributed to this source.

In the conditions just mentioned there are no great circulatory disturbances, but there is a tendency to change in the structure of the arterial walls. As a result of this weakened state, the lumen expands and a larger amount of blood flows through the artery, but its rapidity is decreased. So with the alveolar process, the arteries and veins inclosed within bony walls have little or no expansion, hence the tissues become more susceptible to poisons and toxins circulating in the blood stream. The decreased flow of the blood stream, in turn, causes new connective tissue to form in the intima of the artery, which thickens this outer coat, making it less elastic and reducing the pulse movements.

Any arteries of the body are liable to become involved, but more particularly those of the extremities and end organs. While puberty changes may produce a severe attack, the condition is more frequently noticed later in life—the later, the more pronounced. Men are more subject to the disease than women owing to the fact that women eliminate much more freely than men, and because they are not often subjected to drug poisons. Coldness of the limbs, hard whip-cord arteries with no pulsation, and, in extreme cases, gangrene of the extremities result. The disease begins in the intima and extends to the other coats of the artery. It may be found in all local inflammations of long standing, especially in the extremities, the alveolar process, and may occur in conditions of vasomotor ataxia, such as are present in Raynaud's disease and allied conditions. Syphilis, tuberculosis, typhoid fever, scurvy, and the condition underlying arterio-capillary fibrotic kidney lesions act at times as predisposing causes. Toxins and autotoxic products of retained waste

may disturb physiologic balance, thus giving the pathologic phase of this disorder sway.

Endarteritis is an inflammation of the internal coat of an artery or capillary, generally of chronic type. Its pathogeny is as follows: In direct contact with the blood streams is the endothelium (a layer of flattened cells); next is the tunica intima, composed of elastic fibers arranged longitudinally; next comes the middle coat, composed of muscular fibers arranged transversely. The outer coat consists of longitudinal connective tissue, which contains the vasa vasorum. In the capillaries, the intima lies in immediate contact with the surrounding tissues, or accompanied by a rudimentary adventitia. In other words, the walls of the capillaries consist of almost nothing but the intima. The capillaries have certain contractility; they contract or dilate without muscular fibers. The veins probably also have a certain amount of contraction and dilatation from irritability of

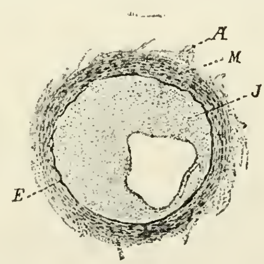


FIG. 79.—ENDARTERITIS OBLITERANS (KAUFMANN).
A, Adventitia. E, Elastic Tissue between Middle Coat and Intima. M, Muscular. J, Thickened Intima.

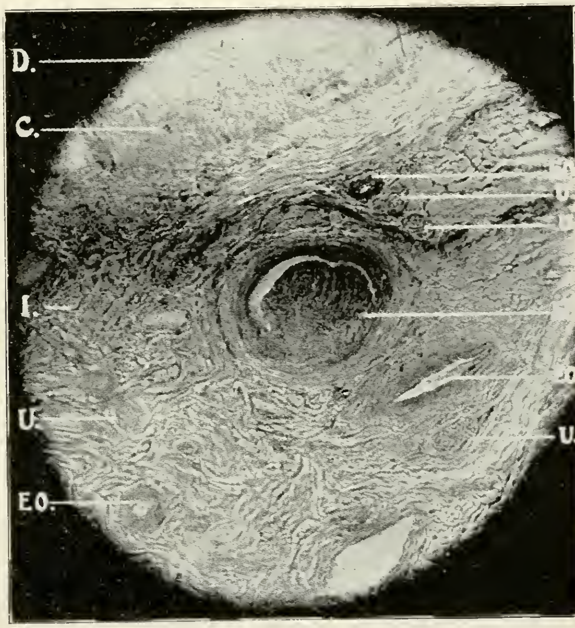
the intima. Each coat of the arteries takes on a special type of inflammation. The causes of endarteritis are numerous.

Inflammation of the intima of the blood vessels may be due to irritation from without or within. When it occurs from without, any local irritation will set up an inflammation which may extend to the outer coats of the capillaries. This produces a marked increase of blood. The vasa vasorum become swollen, the white blood corpuscles crowd into the terminal capillaries and migrate into the extra vascular space. Rapid proliferation of the round-cell elements takes place. The walls of the vessels become thickened. Owing to the projecting intervals of the intima, the caliber of the blood vessels diminishes (Fig. 79).

Irritation occurring from within, results either from trophic changes in the system from direct irritation from toxæmias, or from both interdependently. Under these circumstances toxins may have an affinity for a certain organ, tissue or part, and produce irritation in the capillaries in a distinct part of the body, or the capillaries through the entire body may become involved. Thus, in typhoid fever, the Peyer's gland in the intestine becomes involved; in scarlet fever, the skin or kidney; in malaria, the liver and spleen; in Bright's disease, the kidney; while in mercurial and lead poisoning and scurvy, the mucous membrane, and especially the gums, become diseased. In many of these conditions, however, before the tissue already irritated becomes involved, the nervous system has become affected. The nervous system may already have become affected from other causes. Thus, locomotor ataxia, traumatic injuries to the spine, paretic dementia, cerebral paralysis, neuroticism and degeneracy, and last, but not least, stomach neurasthenia. The poison in the blood, together with the diseased peripheral nerves, produce irritation and inflammation of the inner coat of the capillaries. If this irritation does not disappear soon after its inception, the inflammation tends to affect the other coats of the blood vessels. Under certain conditions, endarteritis may, however, never involve the other coats of the vessels. When irritation of the inner coat of the capillaries takes place, proliferation of the endothelium occurs. This inflammatory growth tends to obstruct the lumen of the vessel. The media may likewise become thickened by an increased connective tissue. The capillaries become obstructed, and finally obliterated. This finally impedes the circulation. Fig. 80 shows such a condition in the scurvy case, elsewhere illustrated.

Irritation may be of less intensity but greater duration, as in cases of syphilis, tuberculosis, scurvy, mercurialism, plumbism (lead poisoning), etc, and the results are then slowly effected. Proliferation of sub-endothelial connective tissue gradually increases until it reaches its limit (endarteritis obliterans). This influence of the proliferation is exerted in addition to that of the round-cell infiltration about the structure.

The recent studies of Hektoen[1] on meningeal tuberculosis demonstrate that tubercle bacilli may penetrate the unbroken endothelial layers of the vessel and stimulate marked proliferation of the sub-endothelial connective tissue. An internal irritant, such as may be produced in the course of any infectious disease or from suboxidation, probably acts upon the endothelium of the walls of the smaller blood vessels in such a way as to permit the escape through the walls first of serum, then of leuco-



X 150. D. D. obj. Zeiss.

FIG. 80.—CROSS SECTION OF PERIDENTAL MEMBRANE, SHOWING ENDARTERITIS OBLITERANS. SCURVY IN MAN.

C, Cementum. D, Dentine. I, Peridental Membrane. U, Nerve Tissue. EO, Endarteritis Obliterans.

cytes, the latter infecting and surrounding the vessels. The effect of the chronic endarteritis is to check the blood supply to the gum tissue. Mercury, lead and other poisons circulating through the blood are forced to remain, hence discoloration of tissue along the gum margin. Interstitial gingivitis, resulting in a slow disturbance of nutrition, produces overgrowth of connective tissue. In all cases of chronic interstitial gingivitis, as shown in the illustration, are the blood vessels thus involved.

[1] American System of the Practice of Medicine, page 119.

Among the predisposing influences which cause this disease are syphilis, tuberculosis, mercurialism, plumbism, brass poisoning, lithæmia, nephritis, gout, rheumatism, alcoholism, scurvy, nervous diseases, pregnancy and old age. Under certain conditions of the system any and all diseases which tend to lower the vitality, producing anæmia, will assist in producing this disease. The direct cause may be resultant overstrain of the blood vessels.

Owing to obliteration of the arterioles in the alveolar process stasis of blood must follow. The detritus from the alveolar process, therefore, must remain in the tissue and collect upon the roots of the teeth.

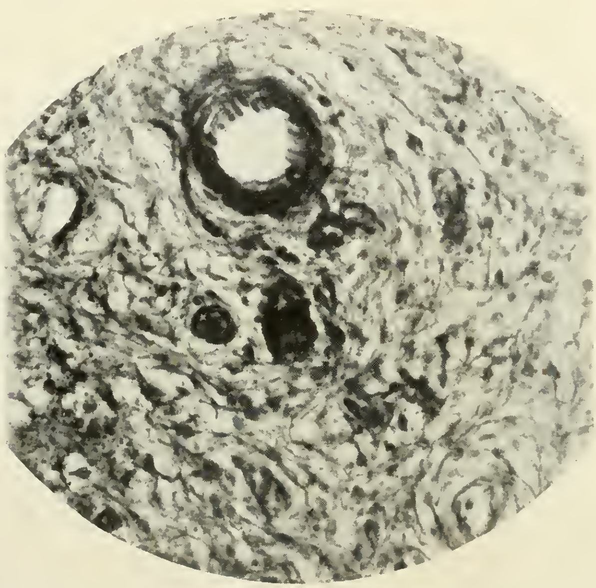


Fig. 81.—Longitudinal Section of Gingival Border, Higher Magnification, Showing Round-Cell Inflammation Extending to the Inner Coat of the Blood Vessel, and also Plasma—Mast Cells.

Endarteritis obliterans and arteriosclerosis of the blood vessel walls in the alveolar process are always observed in connection with both local and constitutional diseases.

No structure affords such a favorable opportunity for the study of endarteritis obliterans and arteriosclerosis as the alveolar process in animals and human, since it can be obtained in quantities at all times and under all conditions. It may be produced in healthy animals by the internal administration of drugs, metals and other poisons.

My researches on this series of experiments were made upon humans, monkeys, and dogs. Decalcification of the alveolar proc-

Fig. 82.—Arteriosclerosis in Tuberculous Monkeys.

ess was made in weak acid solution and prepared for the microscope in the usual way.

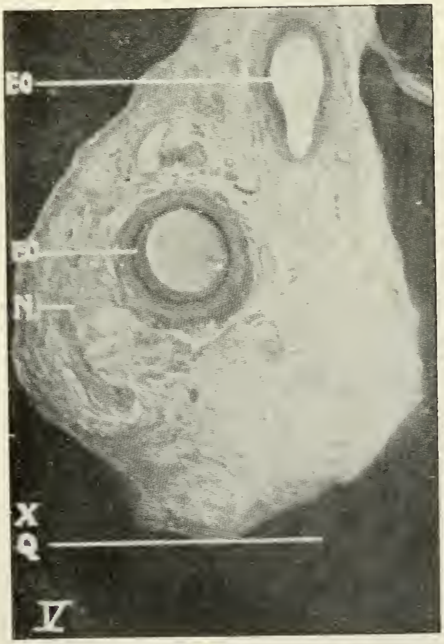


Fig. 83.—Transverse Section of Alveolar Process, Chronic Inflammation Extending Throughout. Dog.

On the administration of drugs, especially mercury or lead, to healthy young dogs, inflammation of the alveolar process with

diseased arterial walls is seen at the end of a month or six weeks. Fig. 81 shows the commencement of the thickening of the intima in a dog. The coats of the arteries are well defined and the inflammatory process has just begun. Examination of the alveolar process of animals or human beings suffering from disease, in which the eliminating organs are not throwing off effete mat-

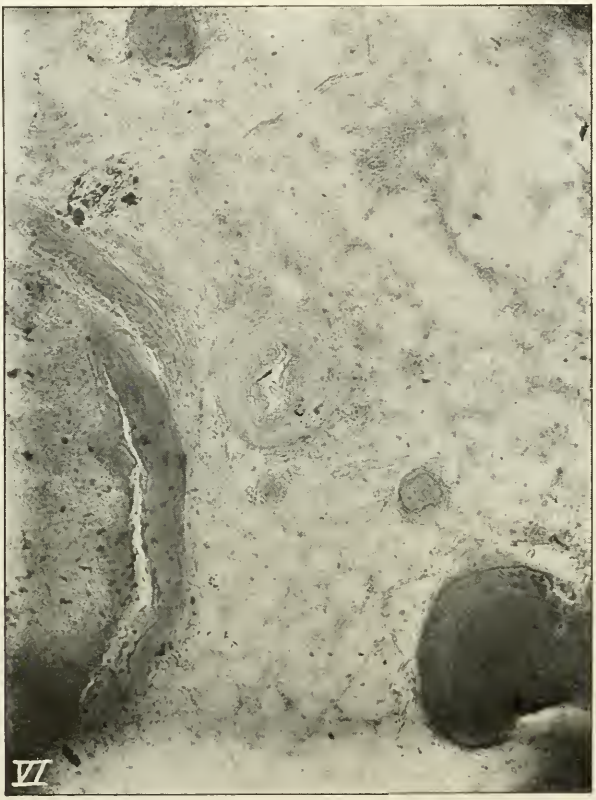


FIG. 84.—ARTERIOSCLEROSIS AND OBLITERANS IN ARTERIES OF A DOG WITH INTERSTITIAL GINGIVITIS.

ter, especially in syphilitic, tuberculous and scorbutic patients, easily reveals this morbid state.

Fig. 82 is a poor illustration of the disease in pregnancy. If such patients are degenerates the process will be exaggerated.

Fig. 83 illustrates endarteritis obliterans in the artery of a dog with interstitial gingivitis.

Fig. 84 is from the alveolar process of a tuberculous monkey.

FIG. 85.—ARTERIOSCLEROSIS FROM MERCURIAL POISONING.

FIG. 86.—ARTERIOSCLEROSIS FROM LEAD POISONING.

Fig. 85 illustrates the closing of three arteries from mercurial poisoning.

Fig. 86 shows endarteritis obliterans with arteriosclerosis in interstitial gingivitis from lead poisoning.

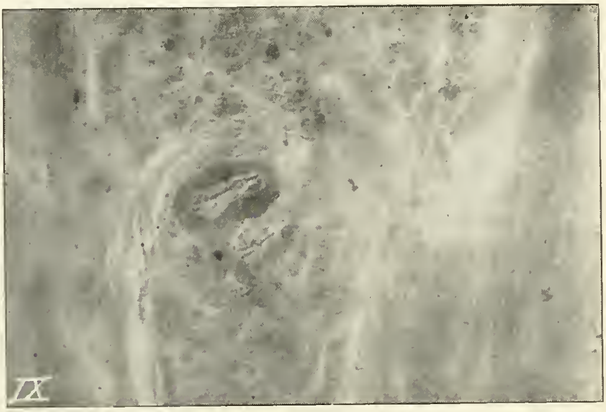


FIG. 87.—ARTERIOSCLEROSIS AND OBLITERANS FROM DIABETES MELLITUS.

Fig. 87 shows arteriosclerosis and endarteritis obliterans in interstitial gingivitis from diabetes mellitus.

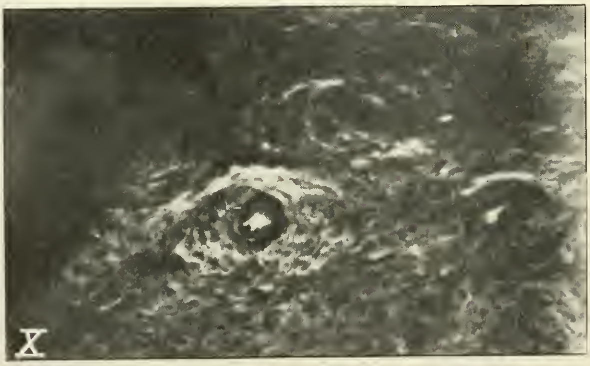


FIG. 88.—ARTERIOSCLEROSIS AND OBLITERANS FROM A SYPHILITIC.

Fig. 88 illustrates arteriosclerosis of three arteries in a syphilitic.

It will be seen from the illustrations that these pathologic conditions in the blood vessels of the alveolar process produce stasis of blood which cuts off the nutrition of the tissues. This, in turn, not only lowers the vitality of the parts, but together with local disturbances causes rapid destruction of the gums, peridental membrane and alveolar process.

NERVE END DEGENERATION.

Since the brain presides over development of the tissues of the body through its trophic and vasomotor systems, it must be as fully developed and normal in construction as possible so that body tissues may develop normally. Pleasure, happiness and laughter aid digestion, while melancholia and grief may retard growth and function and produce tropho-neuroses. An unstable nervous system produces unstable tissues, i. e., either excessive or arrested.

While the nervous system has other special functions, the one great object is that of regulating growth and repair. As Marinesco has shown, this function resides even in the neuron or nerve unit. Growth and repair are regulated through the trophic and vasomotor systems. In the domain of bone growth, trophic nerve anomalies were first observed. Brown-Sequard demonstrated anomalies in tabetic joints of sufferers from locomotor ataxia and later similar states were observed in the jaws. Another allied neurosis, paretic dementia, presents similar trophic disturbances, as Kiernan pointed out thirty-five years ago.[2]

Among these tropho-neuroses is one characterized by loosening and falling out of the teeth by alveolar resorption, gingival ulceration and perforation, with at times maxillary necrosis. This condition has long been recognized by alienists and neurologists as causing that loss of the teeth which occurs in paretic dementia, locomotor ataxia and diabetes. This function of the trophic nerves, as I have elsewhere shown, has received but little attention from dentists, albeit its influence has been recognized in dental pathology in connection with the great neuroses in which gum disorder occurs, followed by a loosening of the teeth.

Degeneration of the peripheral nerves due to interruption of the connection with the central nervous system was first shown by Nasse and Valentine in 1839. Not until 1850, however, was a thorough study made of nerve degeneration by Waller, the pathology of which is now known by his name. Wallerian degeneration implies change in the terminal ends of the peripheral nerves after they have been cut, which consists in coagulation or

[2]Journal of Nervous and Mental Diseases, 1878.

breaking up of the myelin sheath, destruction of the axis cylinder, the neurilemma with its nuclei remaining for some time preserved. If a sensory nerve be cut through peripheral to the spinal ganglion complete degeneration ensues.

Similar experiments showed that if the dorsal root of a spinal nerve be cut through at a point between the ganglion and the spinal cord the portion of the nerve attached to the ganglion did not undergo the typical degeneration, while the portion still connected with the cord showed the characteristic degeneration phenomena which could be traced throughout the whole course of its constituent fibers in the dorsal funiculi of the cord. The cells of the spinal ganglia have therefore been looked upon as trophic centers for the peripheral sensory nerves and their intramedullary continuations.

Similar degenerations in the domain of the central nervous system likewise occur; secondary descending degeneration of the pyramidal tract, established by Turck, and ascending secondary degeneration in the spinal cord after transverse lesion being analagous.

Converting then, as Barker[3] remarks, the Wallerian doctrine into terms of the neuron concept, the following general law may be laid down: "Whenever it has suffered a solution of continuity, with severing of its connection with the cell body and dentrites of the neuron to which it belongs, the axon, together with the myalin sheath covering it, undergoes in the part distal to the lesion acute and complete degeneration. This degeneration includes not only the main axon, but also its terminals, together with the collaterals and their terminals connected with it."

Some investigations have shown that the slightest injuries to nerve cells or neura will give rise to easily demonstrable degenerative lesions in other parts of the cell. The most significant instance is in lateral sclerosis, where the pyramidal motor cells of the cortex show no marked lesions, though the most distal portions of the nerve fibers arising from them have gradually degenerated.

In some peripheral nerve diseases, according to Strumpell,

[3] The Nervous System. Barker.

the degeneration of the distal portion of the axones may be due to direct action of toxins exerting a deleterious influence upon the cell body or the whole neuron. In Wollenberg's opinion the primary type of disease of the sensory neura in tabes is of this kind.

As Sidney Kuh[4] has shown, in some of the toxic forms, as for instance in neuritis due to poisoning with lead and arsenic, the cells of the spinal cord as well as those of the spinal ganglia and brain may be diseased, and according to the neuron theory the toxic substances attack these cells before the nerve fiber itself is altered. Such an assumption explains why pronounced degeneration of peripheral nerves may occur without causing any appreciable symptoms. Toxins and intoxications will produce the same results, especially in those nerves extending into and through the alveolar process. Pitres and Vaillard first showed that after typhoid fever, many nerve fibers are found degenerated, in cases in which, during life, symptoms of neuritis were absent. The same observers found like states in the nerves of those who had died from tuberculosis. Later observations have extended these states to such diseases as diphtheria, syphilis, alcoholism, carcinoma, inanition, marasmus, arteriosclerosis and leprosy; in the so-called rheumatic neuritis of the facial nerve and to inflammation due to articular rheumatism, gout, puerperal infection, tuberculosis, etc.

The method of cell poisoning has been observed in other intoxications. Certain groups of neura are more susceptible than others to a given toxication. The same group of nerve cells in two individuals may react very differently to similar doses of the same poison. Syphilitic toxin shows a decided preference for certain parts of the cerebral cortex, other areas being less affected. The nerve endings in all parts of the body are markedly involved, especially those in and about the teeth. Peripheral nerve degeneration results where the blood current or the nerves themselves are involved from faulty metabolism, etc.

Nerve lesions more readily result where nerves are confined within restricted walls of transitory structures where the pulp

---

[4] American Medicine, Vol. III, No. 21, pp. 865, 868.

has degenerated, especially in cases of hypercementosis of the root. When degeneration of the peripheral nerves in the pulp takes place there may at first be pain, continuously perceptible to the patient or absent except under manipulation or replaced by analgesia. In most cases there is analgesia, owing to the peculiar anatomic construction of the tooth and nerve degeneration. There is loss of function. The same condition exists in the alveolar process when diseases or intoxications occur, the junction of the peripheral nerves is destroyed, resistance is lowered, disease of the process and peridental membrane results.

CHAPTER XXV.

ABSORPTION OF THE ALVEOLAR PROCESS AND CALCIC DEPOSITS UPON THE ROOTS OF THE TEETH.

Absorption of the alveolar process is the result of irritation, resultant malnutrition, and subsequent inflammation. The osteoblasts and osteoclasts are ever present to build up and tear down bone structure on the slightest provocation. Hypertrophy (building up of bone tissue) is the result of intermittent pressure, and atrophy, or absorption of bone, is due to constant irritation and pressure. As has been elsewhere shown, from its transitory nature the alveolar process is unusually susceptible to these influences. The causes of absorption are loss of teeth by extraction, undue pressure upon one or more teeth from improper articulation (Bonwill), wedging and irregularity correction, heat under artificial dentures, and interstitial gingivitis of local and constitutional origin.

According to Kaufmann, lacunar absorption is the most common type. This may be true in morbid anatomy of bone tissue generally, but it is not true of absorption of the alveolar process. On an examination of hundreds of slides prepared from canine and human jaws (of which characteristic types are illustrated), by far the most common form of absorption was found to be halisteresis. Perforating canal absorption, which Kaufmann has "occasionally met with," is certainly very common, while lacunar absorption holds third position. This order of absorption is accounted for by the fact that where structures are transitory, halisteresis, as quickest method, follows by the law of the survival of the fittest. For the same reason perforating canal absorption should stand second. The blood vessels of von Ebner being most numerous, although considered smaller, would naturally be the second tissue involved. As in interstitial gingivitis, absorption of the alveolar process is invariably due to inflammation, halisteresis apparently starts at

the larger Haversian canals from which this form of absorption invariably originates.

Interstitial gingivitis extends to the alveolar process through the periosteum as well as the peridental membrane (not, as dentists usually believe, by way of the peridental membrane alone). This is demonstrated by the illustrations. The entire alveolar process thus becomes involved. The products of inflammation extend through the Haversian canals (a path obviously evident in pathologic illustrations), setting in action the three forms of absorption as elsewhere illustrated.

Halisteresis Ossium (άολος of salt, στερλοις deprivation) or decalcification, is that process of absorption wherein solution of the lime salts first takes place, while the cartilage or matrix remains for the time undisturbed.

Solution of the lime salts begins at the periphery of the Haversian canal and advances toward the center of the trabeculæ. This absorption follows, as a rule, the bone layers. Bone centers are, therefore, usually the last to be absorbed. Frequently decalcification becomes complete; nothing remaining but the organic matrix or cartilage. Finally, this is also entirely destroyed. As the osteoblasts occur in the matrix or cartilage, it is not difficult to understand that absorption may extend far, yet restoration of the alveolar process may occur. After destruction of the matrix such a restoration is impossible. New fibrous tissue may be partly restored, but it is doubtful if the alveolar process can be.

Both Ziegler [1] and Kaufmann [2] divide osteomalacia into senile and juvenile. The latter occurs most frequently during pregnancy. In senile absorption, after a certain period, the entire skeleton is affected. The condition begins most frequently in the "vertebræ and thorax; later extending to the extremities." In pregnancy "the pelvic bones are first involved, the process then extends to the other bones." It is singular that the alveolar process should have been so much neglected by pathologists, since, in both states, the alveolar process becomes involved long before the bones of the body.

---

[1] Special Pathological Anatomy, page 151.

[2] Pathologische Anatomie.

This is due to three reasons: first, to trophic changes; second, to the alveolar process being a transitory structure; and third, to improper care of the gums at these periods.

Osteomalacia occurs in the alveolar process much earlier than at the so-called "senile" period. It is found at twenty, or even earlier, and has been termed juvenile osteomalacia. At any period beyond that year, it occurs probably from the prematurely senile states of which precocity is a type. The lost tissue is regained often after confinement in the "pregnancy" type, but is never regained in the senile.

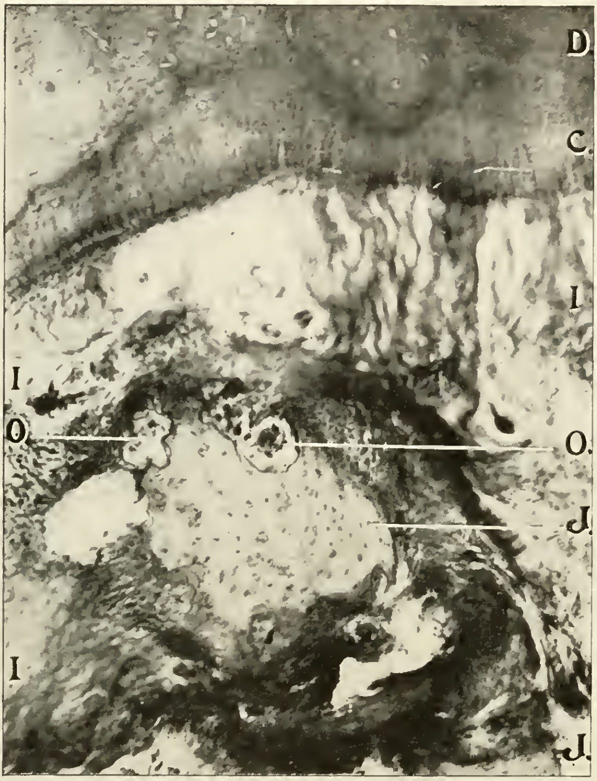
The causes which produce morbid decalcification are not thoroughly determined. Some believe it to be due to lactic acid in the system, others attribute it to an increased amount of carbonic acid in the blood. Eisenhart believes it to be due to a want of alkalinity of the blood, while von Recklinghausen charges it to a local irritation of the vascular mechanism of the bones. It would seem, from examinations already cited, that, so far as the alveolar process is concerned, local irritation from biochemic changes in the blood, as suggested by von Recklinghausen, is the chief cause. Premature absorption of the alveolar process accompanies the movement of the teeth in their correction or in rapid wedging. Frequently the alveolar process is never fully restored, thus producing a predisposing factor for future disease.

Premature absorption, or osteomalacia of the alveolar process, is easily recognized. A shrinking of the gums and alveolar process exposing the necks of the teeth is very conspicuous. Frequently the gums and mucous membrane covering the alveolar process are quite red (this is very noticeable in dogs), and a thinning of the alveolar process over and between the roots of the teeth. The process of one tooth only may become involved; again the process over two, or the whole jaw, and again both jaws become affected.

When osteomalacia occurs, either of pregnancy or senile type, although the tissues be seemingly restored to health, structural change has taken place to such an extent that it ever remains a predisposing factor to interstitial gingivitis.

In Fig. 41 may be seen the blood vessels of von Ebner.

These blood vessels are also to be observed in Fig. 61. They are very common in the alveolar process and, according to Volkmann, are the source of the perforating canals which bear his name.



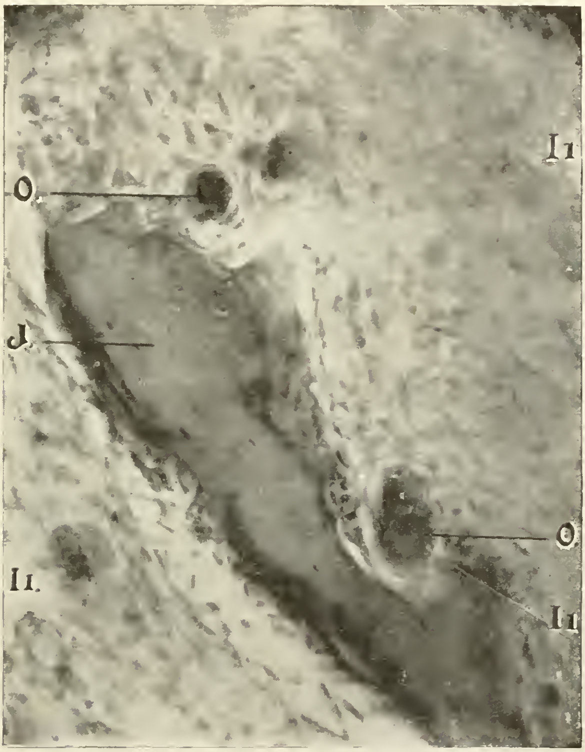
X 50. ½-inch obj. No. Oc.

Fig. 89.—Cross Section of Tooth, Alveolar Process and Peridental Membrane, Showing Lacunar Absorption. Man.

C, Cementum. D, Dentine. I, Peridental Membrane. J, Alveolar Process. O, Lacunar Absorption.

These canals run in all directions. After absorption has gone on to form medullary spaces, these canals penetrate through the trabeculæ from one space to the other (Fig. 43). The position of this type of absorption in the order of frequency comes from the fact that, in this disease, absorption is almost entirely due to inflammation; hence the blood vessels are the first to become involved. Those entering the Haversian canals, being the larger, are first affected, and hence halisteresis naturally precedes.

When irritation takes place in a nerve or part of bone which is about to be absorbed, multinuclear cells arise at the border in the periosteum and peridental membrane. They attach themselves to the surface of the bony trabeculæ. According to Sudduth, "the absorber and the absorbed must be in touch with each



X 300. No. 2, projection ocular. D. D. obj. Zeiss.
FIG. 90.—SECTION OF PERIDENTAL MEMBRANE, SHOWING LACUNAR ABSORPTION IN DOG. J, Alveolar Process. O, Lacunar Absorption. I[1], Inflamed Peridental Membrane.

other." Kolliker has named these cells "Osteoclasts," which term has come into general use. Very soon after these cells make their appearance, cavities are seen in the bone tissue. These cavities are called Howship's lacunæ. Lacunar absorption, as elsewhere shown, takes place as a result of irritation and overstimulation. Fig. 89 shows a cross section of the end of one of the buccal roots of Fig. 91. As will be observed, this tooth was held in place by two buccal roots. As much resistance was required of these two roots as was formerly required of three.

Irritation due to excessive force in mastication was causing absorption. Round-cell inflammation is not present in the peridental membrane. The irritation may be continued until inflammation sets in and until the bone is entirely absorbed, as noticed in Fig. 90. Small round-cell inflammation is quite noticeable in the surrounding tissue.

Sometimes these lacunæ may be seen extending along the entire length of bone. As many as thirty-seven may be counted in some fields (Fig. 42). Lacunar absorption frequently so extends through the Haversian canals as to cut off pieces of the alveolar process. A casual glance at Fig. 89 demonstrates this. This figure could be multiplied many times from other slides. These frequently come away with the peridental membrane when the tooth is extracted. This is often noticed in removing loose teeth due to interstitial gingivitis. By passing the finger over the surface of the root, the rough pieces of bone may be easily felt.

Aside from the forms of absorption already noted, absorption of the alveolar process is often seen, the result of neuropathic lesions. Paretic dementia, diseases of the spinal cord, low forms of inflammation, general debility and traumatism, together with unhygienic conditions of the mouth, are fruitful sources of interstitial gingivitis and absorption of the alveolar process. Absorption of the alveolar process takes place also in diathetic diseases in which the nervous system has been involved (syphilis, scurvy, lithæmia, etc.).

CALCIC DEPOSITS.

There are many instances in which interstitial gingivitis takes place, with absorption of the alveolar process and exfoliation of the teeth, without calcic deposits. In such cases the blood is charged with only sufficient lime salts for the nourishment of the body. The waste products are carried off with the excreta. In absorption of the alveolar process, inflammation does not seem to extend to the capillaries, the result of which is, this waste material is carried into the circulation. In this way, calcic material does not collect in the fluids and upon the teeth. In those cases in which pus is not present (there being a lessened amount of carbonic acid) calcic deposits rarely take place. The

percentage of teeth so found, however, is not so large as those with deposits.

Examination, by a magnifying glass, of a recently extracted tooth (with the root covered with serumal deposits) shows the lime deposited in a manner resembling that of stalactite formation. The deposits often stand out distinctly independent of each other (Fig. 91). This condition is due to deposits from the blood, resultant on biochemic changes in the inflamed tissues. Blood stasis occurs in the gum tissue, fibrous tissue of the perios-

FIG. 91.—PALATINE ROOT OF A MOLAR TOOTH SHOWING CALCIC DEPOSITS.

teum, peridental membrane and alveolar process, through which last much of the blood circulates. This stasis may be consequent upon conditions varying from simple inflammation to disease of the endothelium, producing endarteritis obliterans.

The blood has become surcharged in all constitutional diseases, but more especially in kidney lesions. Deposits occur in the fluids and upon the roots of the teeth. Frequently the deposit is found only on one side or only at one particular spot on the side of the root; again at the apex, when the pulp is destroyed. It may encircle the root. The inflammatory process

may therefore be circumscribed as to area or the whole tissue may be involved. The deposit is circumscribed in the area of inflammation. The calcareous matter absorbed from the alveolar process in the immediate vicinity of the root is soon deposited upon the root or roots because of the impeded circulation.[3] "Ossification, as has been well remarked, is an active development in which the tissues are abundantly supplied with blood. There is a rapid cell proliferation, and the calcareous matter forms an intimate and permanent union with the tissues. Calcification, on the other hand, is passive, and indicates an impaired vitality. Calcification begins as a rule in the interstitial tissue. In regard to the origin of the calcareous salts, it is generally believed that they come more or less immediately from the blood, although Rokitansky supposes that they were formed by a metamorphosis of the tissues involved."

Calcification is due to two varieties of causes: general and local. The former are dependent upon changes in the blood or its circulation, due, for example, to disease or senile change. In composition the blood may be so altered as to contain an abnormal amount of calcareous matter. This effect is most commonly produced by absorption of lime salts from osseous tissues which are the seat of extensive caries, osseous cancer, osteosarcoma or osteomalacia. The calcareous matter thus taken up is conveyed to other and often remote parts and there deposited, constituting the "metastatic calcification" of Virchow. Küttner, of St. Petersburg, has observed a rapid calcification of nearly all of the small arteries as a result of caries involving the dorsal and lumbar vertebræ in a nineteen-year-old boy. Virchow has observed a case in which, as a result of bone cancer (affecting nearly all of the larger bones, particularly the borders of the vertebræ and the skull), the calix and pelvis of the kidneys, the lungs, parenchyma, and the stomach mucous membrane were calcified.

Circulation of the blood may be retarded and thus favor precipitation of calcareous matter normally held in solution. To this is chiefly due the frequency of calcareous degeneration from general loss of vitality.

---

[3] Wood's Handbook of Medical Sciences, Vol. 1, page 743.

Calcification rarely, if ever, depends upon general causes alone. There is, as a rule, a local influence. Very often this is due to pre-existing chronic inflammation. Old accumulations of pus and exudates are exceedingly prone to calcification. The deposit frequently occurs also in fibrous walls surrounding the accumulation. A mere loss of function predisposes to calcification. Such is the case in and about the tissue of the alveolar process. The decalcified material from the alveolar process collects in the soft tissues as well as upon the roots. In his paper George T. Carpenter[4] asks the question: Can a tissue be absorbed and still remain as debris in the pocket? Such is the condition found, and this can be easily proven. Take the contents of a pocket and dissolve it in hydrochloric acid, add three times its bulk of water, to this add ammonia, which will precipitate the phosphate and the calcium. The same results may be obtained by rinsing a freshly extracted tooth of a pyorrhœa case in cold water. With a stiff brush remove the accumulation and place it in a test tube, add hydrochloric acid and more water if necessary. To this add a solution of ammonia and the lime salts are precipitated.

Roots of teeth that have become entirely denuded of peridental membrane and bathed in pus accumulate large quantities of calcic deposits direct from the absorption of the alveolar process.

Difference of opinion exists as to the nature of the process immediately involved in precipitation of lime salts. The simplest and seemingly most logical explanation is that the process is similar to that involved in the formation of stalactites. A certain amount of calcareous matter is a normal constituent of the blood. Herein it is held in solution by carbonic acid, always present in sufficient quantity for this purpose. When the circulation is impeded the free carbonic acid (because of its great diffusibility) is readily absorbed by the tissues or goes to form new compounds, necessitating a precipitation of the calcareous matter. Calcareous matter may be deposited in either a fibrous or fluid matrix. It shows a preference for newly formed fibrous tissue, particularly when this is associated with old tissue under-

[4] Some Points on the Etiology, Pathology and Treatment of Persistent Pyorrhœa Alveolaris.

going fatty degeneration and absorption. In a fibrous matrix the infiltration usually begins in the intercellular substance, but may involve the cellular elements at a later period. In a fluid matrix (like pus) the granules are frequently deposited primarily within the cells. The process may advance slowly or rapidly. When local causes exert the chief influence it is more limited in area of invasion than when there is a general factor in its production, as in the metastatic forms.

From research it has been shown that calcic deposits (other than tartar) may be due, in a limited degree, to a direct deposit from the blood vessels (serumal deposits of Ingersoll) while the greater collection upon the roots of the teeth and in the fluid contents of alveolar and peridental abscesses, is the deposit of the absorbed alveolar process. Analysis of the deposits and of the alveolar process as observed in Chapter VII, shows a close similarity between the two.

CHAPTER XXVI.

PYORRHŒA ALVEOLARIS.

I have shown how inflammation of the alveolar process might be caused by mechanical or local irritation and substances within the organism, without the aid of external infection, namely, irritants in the blood stream. In the last named group are to be included the drug and metal poisons, poisonous gases, etc., auto-intoxication and metabolic disturbances affecting the coats of the blood vessels. The alveolar process is more easily affected by these irritants which may set up inflammation (interstitial gingivitis) in a particular locality and remain there, or it may spread and the entire process become involved. The alveolar process may be destroyed by interstitial gingivitis; it is only necessary that there should be a low form of inflammation taking place in and about the arteries and capillaries to produce absorption of bone. This is what occurs in fully ninety per cent of patients.

When the inflammatory exudate is made up of leucocytes, there is produced within the tissue small round-cell infiltration which becomes so thick as to obscure the tissue. When the leucocytes are in large numbers upon the surface of the mucous membrane about the cervical margin of the alveolar process their appearance on the inflamed surface is that of a white fluid called pus. Owing to the tortuous position of the blood vessels in the alveolar process, the thinnest part being at the gingival border, the inflammatory process usually begins at that point. The pus germs collect at the border, stasis of blood generally being greatest at that locality. This leads to a superficial loss of substance and is known as ulceration or purulent catarrh. When the leucocytes collect in large numbers, within the tissue, and are followed by liquefaction and dissolution, it is called an abscess. These various infections are termed pyorrhœa alveolaris, alveolar abscess or peridental abscess, according to the nature and location of the infection.

Pus infection due to interstitial gingivitis, whether it proceeds from the ulcerated surface or deep down in the interstitial tissues from an abscess, whether it discharges between the gum and root of the tooth or upon the surface of the jaw, must be considered PYORRHŒA ALVEOLARIS, since the source of the pus is always in connection with the peridental membrane lining the socket of the alveolus. PYORRHŒA ALVEOLARIS, therefore, constitutes the second part of this study. About ten per cent of the patients visiting the specialist are thus infected.

Recovery from interstitial gingivitis and return to normal conditions without change in structure is called restoration. Should the damage be extensive, and accumulations of cell and liquid exudate so press upon the tissues as to extinguish their vitality, ordinary restoration is impossible. This is also true when the inflammation is more decided and persistent. This inflammation may extend throughout the tissue. The tissues may be in a favorable condition for infection, yet the mouth and blood vessels be free from pus germs. The tissues are often invaded, however, by micro-organisms, resulting in suppuration. Interstitial gingivitis, with pus infection in and about the alveolar process, resembles suppuration elsewhere in the body.

Suppuration (due to pyogenic cocci) is the usual termination of infective inflammation. Healthy gum tissue is intolerant of bacteria, and will resist the invasion of micro-organisms. When inflammation takes place, the diseased part is unable to resist them. Lowered vitality of tissue is a fruitful source of infection and suppuration. Since, as Miller [1] has shown, pus germs are found in almost every mouth, infection is a very probable outcome of gingivitis.

The organisms most frequently producing pus are the staphylococcus pyogenes aureus, and albus. These have a tendency to accumulate in groups. When they collect at a given point in the tissue, suppuration results. The streptococci (occasionally present in the mouth) do not as a rule produce local suppuration, but spread through the tissue by way of the lymphatics and blood vessels, and eventually give rise to abscess. The delicate reticulum of the blood vessels found in the Haversian canals is a

[1] Micro-Organisms of the Human Mouth.

convenient lodging place for swarms of bacteria, owing to the slowness of the blood current and the tortuous course of the blood channels. When [2] the circulation has been impeded or arrested by an extravasation of blood or congestion of a part, the conditions are favorable for intravascular infection if organisms happen to be circulating in the blood at the time. As we have seen, micro-organisms may from time to time be found in the circulation, particularly in individuals of feeble constitution. The anatomic nature of the part will therefore determine suppuration in certain localities.

In whatever part or tissue the change may occur, the process is the same. The original structures disintegrate. Their place is taken by a closely packed crowd of migrated leucocytes. Should the cause continue to act, the process culminates in the formation of pus. The migrated cells cut off from proper nutrition by pressure are exposed to the injurious action of micro-organisms. The central cells of the group degenerate from want of nutrition or die from direct action of the irritation. The intercellular substance softens, and the liquid exudate from the surrounding parts mingles with the broken-down tissue to form an abscess.

As I have shown, foci of infection and intense inflammation, to the point of degeneration and liquefaction, occur in almost every locality within the peridental membrane, periosteum and what was originally alveolar process. These abscesses are just as likely to point upon the surface of the gum as on the inner surface next to the root of the tooth.

Abscesses in and about the alveolar process (other than those due to dead pulps) are very common. This is due first to the unstable condition of the structures, and second to the ready access of pus germs through the inflamed gums and peridental membrane. Those most susceptible to infection are patients who are anæmic and below par in vitality, and whose gums have become inflamed either from local or constitutional causes. Especially is this the case in those who have osteomalacia where the gums have receded quite a distance from the necks of the teeth. Pus germs collect at the necks of the teeth, infect the raw

[2] American Text-Book of Surgery.

inflamed surfaces of the epithelial layer, and entering the circulation are carried into the deeper structures. Intense inflammation results. Abscesses form, discharging their contents upon the surface. Pus germs also enter the deeper structures through exposed pulps.

Two cases of interest in this connection occurred recently in practice. An active business man, fifty-five years of age, presented himself with an abscess over the buccal roots of the left superior second molar. There were no dead pulps in any of the teeth upon that side of the jaw. Absorption of the alveolar process and contraction of the gums had occurred around all the teeth. He had been overworked and was nervously exhausted. Five years ago cataracts were removed from both eyes. He is exceedingly sensitive to pain. Examination of blood revealed slight anæmia. On examination of urine, other than a specific gravity of 1028, it was found normal. The abscess was lanced and cavity cleansed. It healed within a week. Subsequently he returned with another abscess over the root of the right superior central incisor. Live pulps were in all the teeth upon this side as far as the second molar. Infection, therefore, must have occurred through the gum and peridental membrane.

The teeth of a lady forty-six years of age were being put in order; after the filling of a cavity she called attention to a space between the second and third superior right molars, and stated food lodged at that point, causing pain and bleeding. The space was cleansed with an excavator and the cavity syringed with warm water and then explored. Absorption of the gums and alveolar process had extended one-half the length of the buccal root. Applications of iodoglycerole were made to reduce the inflammation. The patient was dismissed with an appointment for further treatment. She returned at the appointed time with an abscess over the palatine root as large as the thumb. The lady had had acute pain from the time she left the office until her return. The parts had become infected with pus germs through the peridental membrane. The pus was collected in a tin tea spoon, from which cultures were obtained and glass slabs smeared for microscopic examination. The pus was examined

by George T. Carpenter for calcic deposits; the usual aseptic precautions having been taken.

Many dentists, ignoring the laws of pathology, insist that intense inflammation in remote parts of the alveolar process is not due to toxins and irritations but is the result of gouty deposits. The utter lack of foundation for this theory must be apparent on the slightest study of pathology.

Ulceration is always located upon the surface of a tissue. When ulceration occurs from contact irritation of the gum margin or by mechanical or chemical means, congestion and œdema result, thickening of the epithelial layer and increased growth of cells. The sub-epithelial tissue becomes inflamed. The process is not unlike that of the formation of an abscess, since the infected tissue resembles part of an abscess wall. In slowness of progress only does ulceration differ from acute inflammation.

Such is the condition of the peridental membrane. When simple gingivitis becomes chronic, the inflammation extends to the surface of the peridental membrane. This is situated at the lower extremity of a cul-de-sac, formed by the gum on the one hand and the tooth on the other. This cavity is filled with foreign material in which decomposition continually occurs. The tissues are thereby constantly irritated. Necrosis occurs at the surface. In the deeper tissues that have become inflamed pus cells also are found. These not only arise from the normal blood vessels in the vicinity, but also from the granulation tissue. The causes of peridental membrane ulceration are disturbances of nutrition, endarteritis obliterans (a disease of the blood vessels due to constitutional diseases, such as syphilis, scurvy, tuberculosis, uric acid and other blood poisons) and starvation of tissue, feeble circulation (as in anæmia) and inflammation. If the ulcerated surface be examined under the microscope, a general thickening of the tissues will be seen. In the papillary layer deposits of blood pigment occur. The surface is covered with granulation tissue. The tissue may, in part, resemble the type of healthy granulation. It is composed of round cells closely packed together and supplied with rich capillary network. Coagulation necrosis from breaking down of granulation tissue may be present.

Pus pockets start with any local irritation which sets up inflammation (interstitial gingivitis) at the gingival border of the gums. The inflammation spreads to the blood vessels of the peridental membrane and alveolar process. Round cell infiltration rapidly takes place and the bone becomes destroyed, beginning at the gum margin or in the peridental membrane and extending toward the apical end of the root or to the mucous membrane of the mouth. The irritation which is confined to narrower areas may become so intense and the inflammatory exudate increase so rapidly that nothing remains except the fibrous tissue which originally held the bone cells, or the tissue becomes entirely lost. The leucocytes now collect in large numbers within the fibrous tissue and liquefaction and disintegration of tissue results, forming pus pockets.

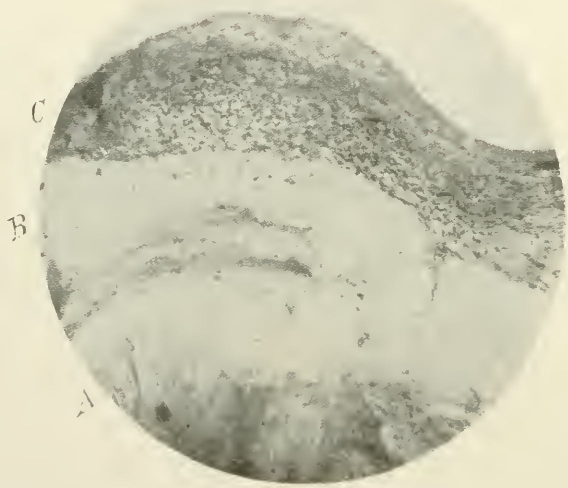


Fig. 92.—Thickening of the Peridental Membrane and Trabeculae (Original).

Alveolar abscess is a term applied to an accumulation of pus at the apical end of the root of a tooth due to death of the dental pulp and other irritations. When death of the pulp occurs, decomposition takes place and gases form in the pulp chamber. The gases expand and an outlet is acquired through the end of the root of the tooth. These gases and other irritations set up inflammation in the peridental membrane, producing an alveolar abscess.

The other irritations may be foreign substances forced through the end of the root or poisons in the organism passing through the blood stream. These irritants set up interstitial gingivitis in the arteries running through the peridental membrane and also into the alveolar process and maxillary bone. Inter-

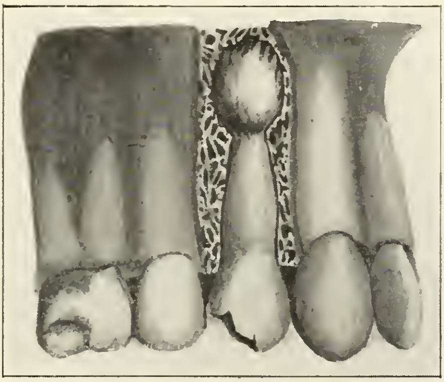


FIG. 93.—SHOWS THE REMOVAL OF THE OUTER PLATE OF BONE AND EXPOSING THE ROOT OF THE TOOTH AND THE ALVEOLAR ABSCESS (ORIGINAL).

stitial gingivitis becomes quite diffused. Bone absorption (halisteresis and Volkmann's canal absorption) immediately takes place and a considerable area of bone about the end of the

FIG. 94.—TOOTH WITH ABSCESS ATTACHED REMOVED FROM THE BONE (ORIGINAL).

root is destroyed, leaving the fibrous tissue (formerly the trabeculæ of the bone) in a thickened condition tightly attached to the end of the root (Fig. 92).

As absorption proceeds, the lime salts in the inflamed area are thus destroyed and the fibrous tissue or trabeculæ become organized (Fig. 93). If the tooth is extracted before liquefaction occurs, the fibrous mass may be removed in situ (Fig. 94). A

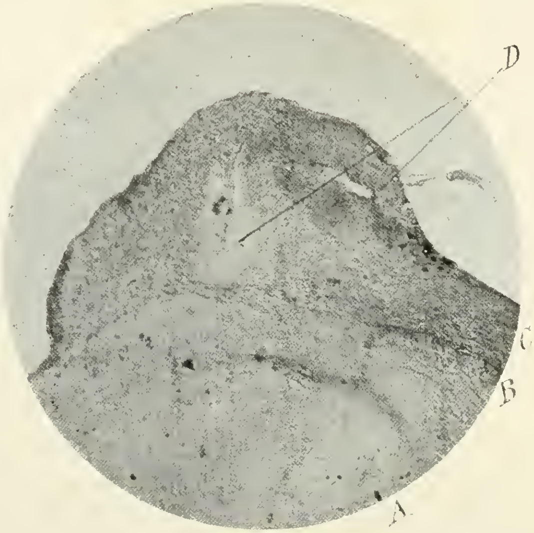


FIG. 95.—MICROSCOPIC ILLUSTRATION OF THE END OF THE ROOT OF THE TOOTH. A, Cementum. B, C, Abscess attached. D, Two Points of Liquefaction. (Original).

low microscopic section of this picture shows the end of the root with fibrous mass attached and degeneration and liquefaction of tissue just commencing at two points near the center of the mass (Fig. 95). A higher magnification showing round-cell

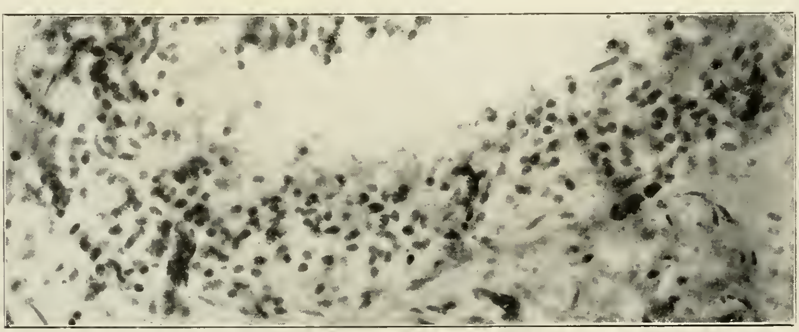


FIG. 96.—MICROSCOPIC ALVEOLAR ABSCESS SAC (ORIGINAL).

infiltration and breaking down of tissue, liquefying into pus is seen in Fig. 96. The pyogenic membrane forming the abscess walls is well shown.

The cause of the irritation producing interstitial gingivitis may be so severe and active that not only is there destruction

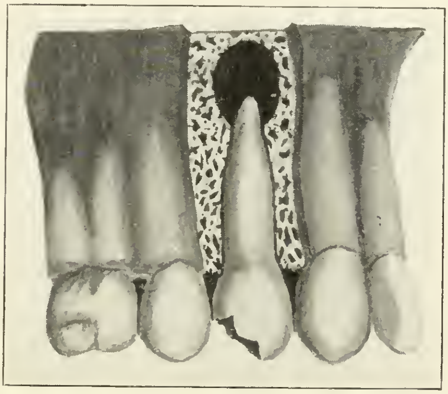


FIG. 97.—TOOTH SHOWING FORMATION AND DESTRUCTION OF ABSCESS WITH CARIOUS CAVITY (ORIGINAL). THE IRRITATION WHICH CAUSED THE INFLAMMATION AND FORMED THE ABSCESS WAS SO GREAT AS TO CAUSE DESTRUCTION OF THE SAC.

of bone, but also of the trabeculæ. Under such conditions the root of the tooth is seen denuded of surrounding tissue (Fig. 97).

FIG. 98.—FOUR ABSCESSES IN THE PERIDENTAL MEMBRANE AND TRABECULAE IN A DIABETIC MAN (ORIGINAL).

In Fig. 98 four pericemental abscesses are seen along the side of the root of the tooth. These are due to the lowered vital-

ity of the individual and infection after interstitial gingivitis has become quite extensive. These abscesses are very common. The tissues about the roots of the teeth become inflamed, infection takes place, abscesses form and discharge. The fistula heals without pain or inconvenience to the patient.

In summing up this chapter on pyorrhœa alveolaris, the reader must not lose sight of the fact that pus infection in no way influences the absorption of the alveolar process and exfoliation of the teeth. It is always the primary inflammatory stage that causes the absorption and the pus infection is only incidental.

CHAPTER XXVII.

CONSTITUTIONAL EFFECTS OF PYORRHŒA ALVEOLARIS.

Every person with pyorrhœa alveolaris has pus germs in the mouth which are constantly being carried into the fauces, stomach and throughout the alimentary canal. That these pus germs under certain conditions are destroyed in the stomach by the gastric juices, to my knowledge, has never been demonstrated. A factor unfavorable to this theory is the non-presence of the hydrochloric acid in the stomach except when food be present, whereas the saliva, laden with pus germs, is continually passing into the stomach and the germs must, without doubt, find their way into other organs, causing grave constitutional states which, in many instances, finally result in death.

The degree of organic infection varies in intensity. The germs passing into the stomach with the food are, on account of the presence of the hydrochloric acid, changed in character, while those passing into the stomach other than during digestion, are more virulent and act with greater intensity upon the intestine and organ walls, since they produce a catarrhal condition, first acute which later becomes chronic. It has been shown repeatedly that many stomach troubles have improved or recovery has been complete when the mouth has been put into an aseptic condition.

The influence of the stage of interstitial gingivitis known as pyorrhœa alveolaris on the system has been discussed by John Fitzgerald,[1] who points out that pyorrhœa alveolaris may act in three different ways in the causation of systemic disease. First, the pus with its multitude of putrefactive organisms and decayed food remnants from the pus pockets is swallowed and either acts locally upon the stomach wall or sets up fermentation of the stomach contents; second, the toxins generated in the mouth may be absorbed by the mucous membrane of the mouth or stomach and thus pass into the general circulation; third, the

[1] Clinical Journal, March 6, 1899.

local conditions of the mouth may favor the growth of pathogenic organisms and thus render the patient more liable to certain infectious disorders, noticeably influenza. The power of pyorrhœa alveolaris to produce aggravation of existing gastric trouble reaches its maximum in cases where there is retention of food residue. This happens when the muscular walls of the stomach are in a state of atony and also when there is some pyloric obstruction which prevents the organ emptying itself. In both these conditions stomach dilatation is eventually produced, with the result that the stomach is never completely emptied. The first condition is a very frequent concomitant of neurasthenia and allied states. It is easy to see how pyorrhœa can at once be predisposed to by neurasthenic states and at the same time increase the neurasthenia by causing gastric trouble through its interference with gastro-intestinal digestion under the conditions mentioned.

Fitzgerald points out that the *bacillus coli communis* is a constant inhabitant of the oral cavity, and, as a rule, seemingly harmless. Under the influences of a culture medium, such as would be furnished by pyorrhœa or an inflammatory state of the gum, this bacteria might, as elsewhere in the mucous membrane, acquire sufficient virulence to produce serious disturbances of the system, such as colitis, dysentery and cholera nostras.

Herschell[2] is of the opinion that many of the chronic indigestions are due to continual absorption of pus into the system from pyorrhœa alveolaris. In these cases he remarks there should be other evidences of the absorption of toxins, such as pigment spots, urticaria, etc.

Within the past few years medical thought has centered around the mouth and its infection as a possible cause of many diseases even far removed from it. The many germs of more or less virulence which have already been isolated in the mouth must of necessity affect mucous surfaces in other parts of the body. Medical researches have shown that diseases like pernicious anæmia, arthritis deformans, all rheumatic states, bacterial endocarditis, headaches, many other obscure conditions,

[2] Indigestion, 1895.

etc., yield more readily to treatment when the mouth has been put into an aseptic condition. Every dentist has experienced the fact that when a poorly nourished patient has had loose teeth extracted, artificial ones substituted and the mouth entirely cleaned up, there will be increase in weight and better health. American and foreign journals are full of experiences of like nature.

Many writers who believe in the theory of the conveyance of pus germs throughout the body have recorded histories of patients who are believed to have been thus infected. Some of these are interesting and here mentioned to show what serious constitutional conditions take place from an unhygienic condition of the mouth and teeth.

There are many affections of the tonsils, neck glands, etc., directly traceable to the septic condition of the mouth and teeth. Especially is this true of school children. Stewart[3] reports 231 cases of tonsil enlargement in which 135 had a caried condition principally of the lower molars on the enlarged tonsil side; 67 had caries and an unhygienic mouth; 16 had no septic condition of the mouth and 15 had caries and a septic state on the opposite side. He also mentions cases of laryngitis which yielded to treatment after putting the mouth in an aseptic state.

It has long been known that nearly all glandular conditions are dependent upon bacterial infection of more or less virulency. Many cases of gland infection have been shown to be directly due to a septic condition of the mouth. A case coming under the author's notice was that of a twenty-three-year-old man whose original mouth condition of pyorrhœa alveolaris was augmented by a local spirrilla infection. After being under treatment for some weeks with no recovery results, he was referred to me. I found the gums, peridental membrane and alveolar process in a severe inflammatory state with pus oozing from the tooth sockets. The neck glands were enlarged on both sides and tender to touch. After the mouth was put in an aseptic state, the glands became their normal size and the tenderness disappeared.

---

[3] Stewart, C. J. Oral Sepsis in its Connection With Throat Diseases. The Lancet, June 25, 1902, p. 1882.

[4] Hunter, W. The Lancet, 1900, 1904.

Hunter [4] reports a case of gastritis which he claims is directly due to infection from the mouth. A sixty-two-year-old woman was suffering with subacute gastritis. There was severe pain and intermittent sickness, so much so that morphia was often resorted to. The illness had been of eight months' duration coupled with loss of weight and great weakness. Examination for cancer of the stomach, abdomen, rectum or uterus revealed nothing. There was continually a bitter taste in the mouth, nausea and loss of appetite. Examination of the vomit found it filled with streptococci, staphylococci and a few bacilli. There were three roots of teeth remaining in the mouth from whose sockets there was a constant flow of pus. With the extraction of the roots the stomach condition was benefited.

It has been a much discussed question among physicians as to whether gastric ulcers are not directly due to a septic condition of the mouth, but convincing proof has not been evidenced since gastric ulcers are known to occur in persons with healthy mouths. In the chronic ulcer type, however, an unclean mouth with pus coming from the tooth sockets is the rule.

Dr. Frank Billings, in a paper on "Chronic Focal Infections and Their Etiologic Relation to Arthritis and Nephritis" [5] speaking of the site of local infection, mentions "the faucial tonsils, abscesses of the gums and alveolar sockets, pyorrhœa alveolaris and septic types of gingivitis may also cause systemic diseases of various types. The systemic results of focal infection are: 1. Chronic arthritis is one of the most common results. 2. Nephritis, both acute and chronic. 3. Cardiovascular degenerations. 4. Chronic neuritis and myalgia (myositis)." He says further, "The studies and experiments embodied in this paper are limited to the arthritides and to subacute and parenchymatous nephritis. Of these, chronic deforming arthritis, commonly known as arthritis deformans, and chronic osteo-arthritis of hypertrophic or atrophic type, comprise the majority of the studies. Next to the arthritides the largest number of cases comprises subacute parenchymatous nephritis and chronic parenchymatous nephritis. The work has been done on private and clinic patients in the Presbyterian Hospital. The bacteriologic

[5] The Illinois Medical Journal, March, 1912.

and histologic studies and the animal experiments have been carried on by Dr. D. J. Davis and by Dr. Homer K. Nicoll."

Ten cases of arthritis and six cases of subacute and chronic parenchymatous nephritis are described. Among this number case Number III is worthy of special mention here.

Case 3.—Mrs. E. W., aged 50 years. I-para. Osteoarthritis chronica, mixed type. Admitted to the Presbyterian Hospital Oct. 16, 1909. For two years there had been swelling, tenderness, pain upon motion and deformity of many of the joints of extremities. Began in feet and hands and extended to larger joints and finally involved cervical spine. The condition was progressive. There was malnutrition, loss of weight from 160 to 120 pounds. For years the patient had been subject to attacks of acute tonsilitis. She had also suffered for years from pyorrhœa alveolaris.

Examination: Poorly nourished, very nervous and irritable. Mouth badly infected, many stumps of carious teeth, some of them loose in the sockets, gums retracted and infected, tonsils large, rough, adherent to pillars of fauces and crypts infected. Breath offensive. Heart, lungs, abdominal organs and pelvic organs normal. There was swelling with some deformity of both ankles, right metatarsophalangeal, both knees, right middle and left fingers, the wrists, and elbows. Some contraction of hamstring muscles prevented complete extension of legs. Both bicep tendons of the arms contracted which prevented extension of the forearm. Twenty-four hours' collection of urine was normal in amount and specific gravity and contained a few hyalin casts. Blood: Hemoglobin, 90 per cent; reds, 4,600,000; whites, 13,400. On Oct. 18, 1909, both faucial tonsils were enucleated by Dr. George E. Shambaugh and one week later the roots of carious teeth were removed by Dr. Frederick Moorhead. From the cut surface of the tonsillar tissues a pure culture of streptococcus was obtained. A rabbit inoculated with a culture suffered from acute multiple arthritis and died in a few days. The streptococcus was regained from the infected joints and from the heart's blood. The patient was permitted to return to her home too soon and did not fully carry out directions as to rest treatment. Some time elapsed before the alveolar processes were

absorbed and the mouth remained sore. On April 3, 1910, she returned to the hospital, where rest treatment was instituted with resulting marked improvement. The patient gained in weight from 129 to 140 pounds. After the return home frequent communications by letter with the patient and her physician have shown that the progress of the disease has entirely stopped. Some of the deformities were so great that one could not expect entire anatomical restoration. The last communication is dated December, 1911, in which the patient says that the strength of her upper extremities and spine is entirely normal. There is some fatigue in the lower extremities after attempting to walk for any great distance, but there is a continued improvement even in this respect.

All the cases cited in this article are of unusual interest because they are based upon researches and actually demonstrate the source of infection and the results of such infection upon animals.

Osler says of the twenty cases of pernicious anæmia which he had under observation in 1909, pyorrhœa alveolaris was present in more than half. Certain types of nephritis are also believed to be due to oral infection.[6]

Zilz [7] reports four cases of cysts at the roots of teeth in which bacteriologic investigations revealed the presence of Much's granula, which he defines as the non-acid-fast form of the tuberculosis germ. The findings are profusely illustrated and the questions discussed are "why it is so difficult to detect acid-fast, Gram-staining bacilli in the gangrenous pulpa," and "why primary tuberculosis starting in carious teeth is not of more common occurrence." He regards Much's granula as merely ordinary acid-fast tubercle bacilli which have lost their acid-fast properties; hence the organic fluids are able to disintegrate them and the vital centers, the granula, escape into surrounding medium. When conditions become more favorable to the bacilli, they may become impregnated again with acid-fast substance and the acid-fast form thus develop again, which in turn may break up anew into granula. His plates show the process dis-

[6] Practice of Medicine, p. 440.
[7] Beiträge zur Klinik der Tuberkulose, Würzburg, Vol. XXII, No. 2, pp. 97-264.

tinctly, the granula causing no tuberculous changes in the dental cyst. The bacilli probably found their way through the blood into the cyst and while there remained latent but regained their virulence when conveyed farther to lymph-nodes or lungs. Inoculation of animals with the granula always gave positive results.

W. Hunter[8] has repeatedly called the attention of the profession to the close relationship existing between the mouth infection and the various forms of anæmia, particularly the pernicious type. He contends that the pus taken into the stomach produces an unhealthy state of that organ and also the intestines, thereby favoring the destruction of the red blood cells.

In all forms of arthritis there is an association in the condition of the mouth. The disease stamps itself indelibly upon the alveolar process. There is gum recession and absorption of the alveolar process of more or less intensity and pus infection.

A case referred to the author for treatment of the mouth is of more than passing interest. The patient, a twenty-seven-year-old woman, unmarried, had been a sufferer from arthritis deformans for many years. For ten years previous to her coming to me she had been unable to leave her home except when carried. The entire osseous system was involved, but more especially the extremities. The hands had become so malformed and stiff that she was unable to pick up or hold anything. The mouth was in frightful shape. There were loose teeth with pyorrhœa alveolaris, absorption of the gums and alveolar process, a number of large cavities, tartar and calcic deposits. After the loose teeth had been extracted, the tartar and calcic deposits removed, the cavities filled, an artificial denture inserted and the pyorrhœa alveolaris treated, the constitutional condition commenced to improve. At the end of three months she could walk with the aid of crutches, in six months with only the use of a cane, and at the end of a year had no use for either. The stiffness in the hands disappeared to such a degree that she could use them. She has now recovered to such an extent that she is able to assist in the housework.

[8] The Lancet, 1900, 1904.

Dr. Kenneth Goadby,[9] of London, England, reports four interesting cases of arthritis which are worthy of consideration at this time. They are as follows:

Case 1.—A girl, aged twenty-two, was attacked somewhat suddenly by swelling of the hands and feet and fever lasting two or three weeks. With the subsidence of the fever the joints did not return to their normal size but remained painful and stiff; walking was almost impossible. The affection was bilateral and the swelling was evidently peri-articular, and to a limited extent affected the synovia of the joints, but no fluid was discovered. Treatment at Bath and a long course of salicylates produced little improvement. There was no family history of rheumatism or of gonorrheal infection and no septic focus was thought to exist. On examination of the mouth, the right upper central incisor was missing, the teeth and gums were apparently quite normal. A closer examination revealed a small sinus leading up to the root of the missing central incisor and a film made from the sinus showed a large number of pus cells loaded with organisms. Cultures were made and an organism was isolated in practically pure culture; the blood tested against this organism gave a very low opsonic as well as a low phagocytic index. A vaccine was prepared and injections given, commencing with ten million dead bacteria. After four injections the sinus was opened under a general anesthetic and was found to lead into a cavity in the bone about the size of a small hazelnut. This was cleared out with a sharp spoon and the lateral incisor also was removed, the cavity extending under its root and invading the periosteum of the tooth. The improvement of the joints which had commenced with the inoculations received a slight temporary setback as the immediate result of the operation, but improvement soon recommenced with continued vaccine therapy and the patient has steadily improved and is almost well.

The second case is that of a man of thirty-eight years. He was suffering from acute pain and swelling in both knees and both feet, ulnar deflection of both hands and acute pain and swelling on the dorsal aspects, fluid and deformity of the left elbow joint and of the left shoulder joint, anæmia and neuras-

[9] The Practitioner (London), January, 1912.

thenia, partly owing to the constant pain and partly toxic. I may note in passing that secondary septic neurasthenia is common in cases of oral infection, probably due to the long-continued infection with small doses of bacterial toxins. The patient had been under all sorts of treatment, residence at Continental and English Spas, had been to the Canary Islands, had taken vast quantities of iodid of potassium, had had massage, electric baths, ionisation and ''Christian Science'' and all with no avail.

His mouth was a veritable gold mine; he had two bridges, two in the upper, two in the lower jaw, and four gold crowns in addition to the bridges; pus was welling up from his gums in all directions. The builder of the bridges told him he could do nothing for him as he had rheumatism in his gums. He was treated by the removal of all the crowns and bridges and by vaccines made from his two organisms. He made a slow but steady recovery and is now enabled to resume his ordinary avocation which he had been obliged to give up for three years previous. Unfortunately the right knee-joint is partially disabled owing to exostosis.

The third case, a man aged forty, in March, 1908, had a sudden attack of pain behind the left ear, progressive stiffness and muscular rheumatism and stiffness of the right shoulder and right hip joints. Ten days later, rigor, temperature 102° F. and evening temperature of 100° F. for two or three weeks which gradually subsided. In the following spring another acute attack with fever, pains in the head and neck, swelling of sterno-mastoid sheath and stiff neck, lasting five weeks. An X-ray photograph of the chest was taken and it was thought the case was one of early tuberculosis; the patient was sent to a sanatorium where he derived no benefit. He was in constant pain, unable to move his head, and had constant attacks of fever at night, the temperature running up to 100° F. and falling to subnormal in the morning. He became wasted, losing more than a stone in weight, was greatly depressed mentally and had to give up his work. On examination in October, 1909, considerable thickening was found in the left shoulder joint and right knee joint. The left sterno-mastoid was thickened in the region of the rectus, capitis posticus and complexus, and thickened tender areas along the

spine process of the cervical and dorsal vertebræ. Hyperesthesia over all cranial nerves. The patient could walk only with difficulty.

The molar teeth had been lost on both sides in both jaws. The patient resented any suggestion that his mouth was at fault, as he had recently seen his dentist who pronounced his gums and teeth quite sound, and the gums appeared normal in color. Careful examination with a fine platinum probe brought to light several deficiencies between the remaining teeth and passing down to bare bone, and microscopically pus was demonstrated. A vaccine was therefore prepared and inoculations were performed. The patient made an uninterrupted recovery, the inoculations were discontinued and a slight relapse took place. The vaccine was therefore continued for a further six months. The patient made a complete recovery.

In the fourth case, a woman aged forty-two, there was a severe general infection of the mouth. All the teeth were loose and copious discharges of pus came from all sockets. There had been chronic progressive arthritis for the last four years associated with occasional acute exacerbations, constant pain in both knee joints which were swollen and thickened, especially the external and lower portions of the capsule; creaking was well marked in the knees and shoulders, with ulnar deflection of the right hand. All teeth were removed and considerable improvement took place. Six months after removal of teeth pain recurred in the right knee and in the shoulders but passed off. A year later, eighteen months after removal of teeth, the pain and stiffness of the knees again recurred together with fusiform swellings and local vasomotor disturbances of the fingers. On examination the gums were found quite healed; there was no inflammation, but small patches of thickening were seen along the outer surfaces of the alveolar process. A puncture was made into these and a pure culture of the streptobacillus malæ was found. Vaccine therapy was instituted and the rheumatoid symptoms rapidly disappeared and two years later no recurrence had taken place.

Every specialist has noted the marked improvement in his patients, some of whom were suffering with obscure ailments,

after having loose teeth removed, pyorrhœa alveolaris treated and cured, gums and mouth placed in an hygienic condition, artificial teeth inserted to fill vacant spaces, and many experiences could be recorded.

Gilmer [10] reports three interesting cases. He says, "Some years ago, a man, a little beyond middle life, consulted me relative to a trivial dental lesion. On making a careful examination of the entire oral cavity, I found several small sinuses discharging pus above the bicuspid teeth on one side of the upper jaw. On exploring these openings with a sharp steel probe a large cavity was discovered in the bone, the result of alveolar abscess, the presence of which was unsuspected by the patient. On inquiring into his physical condition I learned that for the past year he had had a cough, his digestion was impaired, and much of the time his temperature was slightly above normal. He had frequently consulted his family physician who examined his heart, lungs, sputum, urine and blood. These gave no clue to the cause of ill health. His appearance indicated a toxemia. I removed several teeth, curetted the abscess in the jaw and followed it by suitable after-treatment. His fever at once subsided, his digestion was soon much improved, his cough was lessened and finally disappeared altogether. Although seemingly his physician had made a careful examination he had overlooked one important factor, the mouth.

"Mrs. C., aged thirty years, noticed the appearance and disappearance at frequent intervals of an erythemic patch about the size of a silver quarter on the skin over the left canine fossa. On examining the mouth for a possible cause for this reddened condition of the skin I found the left lateral incisor pulpless. There was no sinus and the tooth had given no trouble. The only evidence of disease found in the mouth was a slight hyperemia of the gum over the lateral incisor root indicated. The radiograph showed a pus cavity in the bone at the end of the root about the size of a large pea. Disinfection of the root did not effect a cure. I made an opening through the labial wall of the alveolar process, excised the end of the root and curetted

[10] The Illinois Medical Journal, March, 1912.

the cavity. The erythemic patch on the cheek disappeared and did not return.

"Mr. S., aged about twenty-five years, was directed to my clinic for the treatment of a chronic abscess in the upper jaw in the vicinity of the incisors and cuspid which had proven intractable to ordinary treatment. His physical condition was much impaired, he was emaciated, his skin was sallow, his cheeks hollow, his conjunctivæ pale, eyes dull, and his lips lacked the color of health. His temperature was slightly above normal. I could elicit no history of any other illness, recent or otherwise. His appearance gave the picture of a toxemia. In this case, likewise, the sharp steel probe revealed a large cavity in the bone, extending from the central incisor to the first molar. On Oct. 20, 1911, I extracted the cuspid tooth and curetted the bone cavity. On Oct. 27th, he returned to the clinic much improved. Nov. 10th, his color was normal, his eyes clear, and he seemed well."

Dr. T. B. Hartzell, of Minneapolis, reports the following four cases:

"Case 1. A typical case of pyorrhœa alveolaris. Patient, male, aged 50 years. Molar and bicuspids in both lower and upper arches freely movable in sockets, having lost the bone to about one-half of the original depth of sockets, pus discharging freely from the sockets about the teeth, temperature normal, specific gravity of urine 1018, no albumen, no sugar. Patient reports a tender area in the stomach wall. Diagnosis, ulcer of the stomach accompanied by chronic dyspepsia. Patient has had treatment for ulcer of the stomach for two years with but temporary benefit. The treatment in this case was first extraction of two of the loose teeth followed by accurate planing of the root surfaces of all the teeth which had lost alveolar process. Absolute cessation of pus flow. Gums resume normal tint and after two months no tenderness in the region of the ulcer, digestion about normal.

"Case 2. Male, aged 48. Chronic pain and tenderness in the masseter muscles of the left side. Tenderness of the sublingual glands and torticollis. Tenderness of left shoulder joint. Ex-

amination of the mouth revealed dead pulp in left lower 8 with free pus discharge from deep pyorrhœa pockets. Left lower 6 and 7 vital pyorrhœa pockets one-third the depth of the root. General pyorrhœa of all the molars and bicuspids on both sides of the mouth. Chronic acid indigestion with constant eructation of gas after the ingestion of food. Treatment, extraction of loose left lower 8. Planing of the root surface of all the teeth affected by pyorrhœa. Pockets were pencilled with tincture of iodin. After two weeks, rheumatic pains in shoulder and tenderness of sublingual glands disappeared. Digestion improved. End of fourth week all inflammatory symptoms contiguous to the teeth absent. Teeth no longer tender on occlusion. Patient has resumed vigorous mastication of food. End of two months all symptoms of dyspepsia absent. This case is typical of a group of five cases in which joint involvements have been present from one to three years which have all disappeared upon the stamping out of oral infections.

"Case 3. Mrs. J., aged about 50, with mild inflammation of the gum margins. Abscess in the region of left upper 2 penetrating the palatal tissues an inch and a quarter. Patient enjoys moderately good health though a constant sufferer from constipation, rheumatism of the arms, wrists and fingers, and of the feet, ankles and knees. Pains sufficiently sharp on rising to cause marked discomfort. Draining of the abscess and treatment of the interstitial gingivitis results after two months in complete freedom from rheumatic pain and also freedom from constipation.

"Case 4. Miss A., aged 26, suffered chronic dyspepsia and neurasthenia. Has been treated for three years in various sanatoria and has also spent two winters in Southern California. Patient presented the winter of 1906. Mentally, much depressed. Physically, very weak. Weight, 86 pounds. No albumen, no sugar. All the teeth exceedingly loose. Deep pockets discharging pus freely. Teeth all extracted except four in the upper and four in the lower jaw. These eight teeth were treated for pyorrhœa and although they were so placed that they were of no value for mastication the patient showed a marked improvement. At the end of one week had gained one pound in

weight although obliged to subsist largely upon liquids. Artificial dentures were placed, and the patient increased in weight steadily at the rate of a pound a week until her normal weight of 106 pounds had been regained, after which time she returned to her home."

Dr. David J. Davis,[11] Pathologist of St. Luke's Hospital, reports the following: "Pyemia, septicemia, meningitis, neuritis, endocarditis, etc., as acute infections originating from alveolar abscesses and infected food about the teeth, are well known occurrences. Chronic generalized or systemic infections are rarer and less commonly recognized. As an illustration I may mention briefly one rather striking case which came under my observation.

"A young man had been suffering for several weeks with symptoms of severe multiple neuritis associated with some anæmia, marked emaciation and slight fever. The joints were not involved and physical examination revealed no heart lesion. For a long time the patient had been troubled with severe pyorrhœa and at the time of examination the gums of the lower jaw were red and swollen, bled easily and on pressure abundant pus exuded from between teeth and gums. Smear and culture examination of this pus revealed, in nearly pure growth, many Gram-positive diplococci resembling pneumococci. The colonies produced a green zone on blood-agar and the organism acidified and coagulated milk and fermented inulin. A blood-culture made by taking several cubic centimeters of blood from the vein at the elbow yielded in each of three inoculated culture-tubes a pure growth of the same diplococcus. In animals this organism was not highly pathogenic. One broth-culture intraperitoneally would kill guinea-pigs in twenty-four to forty-eight hours. Two rabbits were inoculated repeatedly with large amounts of culture without manifesting serious or fatal effects. Apparently these diplococci are identical with the cocci often found in endocarditis and called endocarditic cocci and they are also probably identical with the diplococcus almost constantly found in the mouth and usually called *Streptococcus viridans* (Schottmüller. I may say, too, that they appear to be the same as the diplococci

[11]Archives of Internal Medicine, April, 1912. Vol. IX, pp. 505-514.

which I have found at times in the tonsillar crypts, especially in cases of endocarditis, and which I will discuss later in the paper. In this particular case the tonsils were carefully examined and appeared to be normal and no suspicious foci of infection other than the teeth could be held accountable for the condition. Unfortunately, the patient did not remain long under observation and the termination is not known."

Dr. Edward E. Rosenow of the Pathological Laboratory, Rush Medical College, says, in a letter to the author, "Clinical observation convinces me that the low grade infections about the teeth, etc., which are looked upon so often as harmless, are far from being so. The evidence is practically conclusive that endocarditis may have its origin in some such infection. In the September number of the Journal of Infectious Diseases there will appear an article on endocarditis in which these points will be taken up. The mechanism of how the organisms which are so common in these infections about the teeth can produce endocarditis is shown experimentally in the rabbit."

At the meeting of the American Medical Association held at Atlantic City in June, 1912, Dr. E. Libman of New York exhibited in the pathological exhibit a series of twenty-two hearts showing bacterial endocarditis due to mouth infection.

Medical literature records many ailments and cures of disease associated with pus infection in the mouth. While every practitioner would logically reason from his every-day experiences that there is a relationship existing between cause and effect, yet more absolute data in the way of research is necessary to show more clearly that such relationship exists. The work of Billings, Rosenow, Davis and others should be confirmed by many more experiments upon animals to verify these facts.

The author is one of those who believes that infections of the mouth are taken into the stomach with every swallow; and that infection of the glands of the neck as well as absorption of pus directly into the blood and carried to all parts of the body, occurs in a large percentage of our patients, because he has cured many thus infected and improved the health of others by putting the mouth in a healthy condition.

CHAPTER XXVIII.

TREATMENT.

The treatment of interstitial gingivitis as a whole is very unsatisfactory both to the operator and the patient. The clinical history of each structure is essentially that of any other disease of the mucous membrane, periosteum and bone tissue. From a microscopic viewpoint, however, as illustrated by the author's researches, the pathologic aspect is quite complicated, since the relation of tooth, peridental membrane, alveolar process and gum tissue has no counterpart in any other part of the body.

The etiology of most of the disease to the dentist is obscure in nearly every person under treatment, as there are constitutional factors involved in connection with the local irritation. The dental specialist not having a medical education and a general knowledge of disease intoxications, the faulty metabolism and lowered resistance are not understood.

There is a general law in medicine laid down many years ago that to treat a disease successfully the cause must always be removed. In those patients in whom the cause is of a constitutional nature, there may or may not be local deposits. The mere cleansing of the tooth roots and local treatment will not always cure such conditions. On the other hand, much harm may be caused (as we have already shown) by local irritation, traumatic lesions, and changes in function which cause the tissue to be readily acted upon by irritants in the blood stream. A large percentage of interstitial gingivitis is influenced or entirely due to constitutional causes. Interstitial gingivitis under such circumstances can never be permanently reduced until the cause has been ascertained and remedied. A very thorough history of the patient, especially age and condition of the urine, should be obtained. Such data will assist the specialist greatly in a clear understanding of his method of procedure.

We have shown that the tooth, so far as this disease is con-

cerned, is a foreign body and the alveolar process is an endo-transitory structure. When disease, therefore, whether due to local or constitutional causes, once affects these structures, even if they be restored to comparative health, they are more easily involved than other structures of the body. When inflammation has once taken place in these tissues, whether the disease be due to local or constitutional causes, the operator may apparently restore these structures to health. If, however, the cause is not removed, whether it be local or constitutional, the disease soon returns as it is of a progressive nature. This necessitates frequent visits of the patient for further treatment. The endo-transitory nature of the alveolar process, after inflammation has once affected the part, naturally causes the disease to become chronic. The obscure constitutional causes and the chronic tendency of the disease soon causes the tooth to become loose and finally exfoliated.

When the disease is taken in hand and continually treated before the bone becomes involved, a fair success may be obtained. I have demonstrated that bone absorption around the teeth as one grows older is a natural process which cannot be arrested to any extent. Toxins and irritations assist greatly, dependent always on the resistance of tissue and strength of the irritant. Neglect on the part of the patient to massage the gums properly twice a day and use a proper gum wash, the irritants in the blood and in the mouth, cause the tissue to soon return to their pathologic state when the bone becomes diseased, owing to its endo-transitory nature. Chronic inflammation once set up, "eternal vigilance" on the part of the operator and patient is the only method that will prevent the disease from progressing with a final destruction of the alveolar process and exfoliation of the teeth.

The disorder responds quickly to treatment at its outset. Later, its complications and the extent of structure involved, render treatment very inefficacious, and always insure loss of the tooth. As the general surgeon's duty is to save life, if need be, at the expense of limb or organ, but to save these last if possible, so the dental surgeon's duty is to remove loose teeth, if need be, for the benefit of the general health, but to save them,

when possible, for the same reason. The patient, therefore, should be told frankly at the outset of interstitial gingivitis, that it is a condition requiring time for its treatment, and should not be given that prognosis too frequently made of quick cure. To such a prognosis many a case of constitutional disorder is due. The dentist is a practitioner of a surgical specialty, not a mere tooth-puller. The surgical side of dentistry has received too much attention, however; the medical or prophylactic too little. Patients are beginning to pay more attention to the prophylaxis of diseases of the teeth and jaws, and need but little encouragement and instruction to see the absolute necessity of early prophylaxis and treatment of interstitial gingivitis. The trend in general medicine is to prophylaxis, and this has undoubtedly so impressed patients as to open the way for dental prophylactic suggestions. Viewing the question from the narrowest standpoint of remuneration, the dentist could not fail to profit by instructions to his patients on prophylaxis. He certainly fails in his duty as the member of a learned profession by not doing this. Furthermore, with the known necessity for prophylaxis, it is an open question whether the failure to inform the patient of the dangers of the incipient disease could not be successfully pleaded as a basis for a malpractice suit.

From the etiology of this disease, the treatment would appear simple and easy.

Early diagnosis is not difficult, since the simple inflammation of the gums is easily recognized by the patient. Bleeding when the toothbrush or toothpick is used can never be mistaken. The dentist with his accomplished eye can readily detect the slightest change in color or puffiness around the necks of the teeth or of the festoons between the teeth. Redness, puffiness and bleeding are pathognomonic of this disease in its incipiency.

Few dentists have, however, given this stage of the disease any thought, albeit they have filled the teeth of their patients from year to year. I have in mind three patients with loose teeth and inflammation extending throughout the peridental membrane and alveolar process, who had been under an old practitioner now retired from practice. The patients had never had the gums treated or even their teeth cleaned. This is not

an uncommon occurrence. The excuse usually made by the dentist is that he cannot get paid for his time. Gingivitis is a disease which the dentist is as much bound to treat and cure as any disease of the mouth and teeth. It is a part of his specialty which should not be ignored. It is claimed that the dental profession is overcrowded. Were this disease treated until the gums were placed in a healthy condition, there would be practice enough for as many more dentists as there are today. The busy dentist of today could attend only to one-half the patients whom he now serves.

The treatment, then, should be prophylactic in its nature, preventive rather than corrective. The disease and treatment is not unlike an inverted pyramid: the farther from the apex or beginning, the more difficult and hopeless the task becomes. Since the teeth have nothing directly to do with this disease, they should be ignored. In the early stages, the gums should receive proper attention. These, like other parts and organs of the body, must be exercised and kept clean to be healthy. The gums should be properly massaged, just as the liver, kidneys or skin are when they are not doing proper work. This can be accomplished by properly made brushes. The ordinary toothbrush is not adapted to the work under discussion. It will brush the teeth but not reach the gums. What is needed is a massage brush that will miss the teeth to a certain extent, but will reach the gums and contract them tight around the teeth,

Fig. 99.—The Author's Gum Massage Brush.

thus preventing the accumulation of foreign substances. The patient should be instructed with this single idea in view, "that the gum margin is to be exercised and stimulated and not the teeth, which must be ignored." A brush, properly made for gum massage (Fig. 99), will do sufficient work upon the teeth with the aid of the floss silk and toothpick. It should have

printed upon the handle, in large letters, "gum massage brush." The handle should be bent a little more than the "prophylactic," so that the end containing the bristles can be brought in contact with the gum, posterior to the central incisors, upper and lower, and around the third molar teeth. There should be a tuft of bristles at the point with a space for the teeth. The tuft should be longer than those on the body of the brush. This tuft will reach the gums at all points inside of the mouth and around the molars. The bristles on the body must have spaces between them, so that when the upward and downward movement is given, the bristles will go between the teeth and reach the gum festoons. The bristles must be medium and hard. The quality of bristles must depend, however, upon the condition of the gums. If they be soft and spongy, the medium may be used. If the processes are heavy and thick, the gums swollen and engorged with blood, hard bristles must be used. Soft bristles (although sometimes recommended) should never be used.

The antique theory that vigorous stimulation is injurious is too much accepted. Barrett,[1] for example, says, "massage of the gums with the ball of the finger and by the frequent use of a rather soft brush should be resorted to." Bœdecker[2] remarks that too frequent application of the toothbrush is sufficient to produce papillary hyperplasia. Tomes[3] says, "in my own experience I have found that frequent and vigorous rubbing of the gums with the finger, shampooing them in fact, has often been productive of great advantage, the patient of course being cautioned not to rub the actual edge; but even on this point there is difference of opinion, for in a recent paper on the subject, rest and the avoidance of all friction is advocated." Dr. Meyer L. Rhein,[4] in introducing the "Prophylactic Toothbrush" to the profession, says, in his article on "Oral Hygiene," each brush comes inclosed in an envelope, upon which are printed directions for the intelligent use thereof, and the following caution: "Never pass the brush across the teeth, as this movement de-

[1] Dental Cosmos, 1883, page 532.
[2] Anatomy and Pathology of the Teeth, page 365.
[3] Dental Surgery, page, 704.
[4] New England Journal of Dentistry, October, 1884.

stroys the delicate membrane which attaches the gum to the teeth, causing recession of the gum, and ultimate loosening and loss of the teeth.'' Citations of this could be multiplied, showing the general impression is that the gums should not be stimulated to any great extent; that the finger, a soft cloth, or a very soft toothbrush alone should be employed. The use of the finger is a superstition which is handed down from generation to generation without the slightest critical analysis. If the advocate of this use would try the experiment, he would see how impossible it would be to bring it in contact with all the tissues of the mouth that are involved in this disease; were it possible, the fingers, cloth and soft toothbrush would not accomplish the desired result.

No brush should be used whose bristles are softer than the medium; very often these, used once or twice and dipped into water or mouth washes, become so soft as to be wholly unfit for use. It is always a good plan to have two brushes to be used on alternate days. In this way one can dry while the other is being used. The general opinion has been that friction upon the gums was detrimental on account of the resultant tendency to absorption of the gums. While this may exceptionally be true, it is not true of a majority. Should milk, arsenic, iron, strychnine or quinine be entirely abolished as remedies because occasionally a person presents untoward effects? If the alveolar process be very thin over the roots of the teeth, especially the cuspids, the patient must be instructed to use the brush so as not to overstimulate these particular parts. In such cases the inner alveolar process and gum tissues may be stimulated with impunity and with the hardest brush. Again, if the chronic interstitial gingivitis be of long standing, or even if chronic gingivitis has been present for some time, stimulation of the brush will cause the gums and mucous membrane to recede until hard, sound, healthy bone structure has been secured. Then absorption for the time being will practically cease. In most cases absorption and contraction of the gum tissue will take place to a more or less marked degree. If absorption of the alveolar process has taken place and the gums are puffy, red and swollen, a disease exists, to be cured, regardless of consequences. The alveolar

process and gums will never return to their original position, but it is a decided advantage to have a healthy mouth, even if the alveolar process and gums have slightly receded.

I have used medium and stiff brushes in my practice for the last thirty years and have failed to see any ill results. For the past sixteen years I have made constant experiments, with the view of securing the proper shape and stiffness of the bristles and have obtained uniform results in gum treatment.

Proper employment of the "gum massage brush" requires skill. Every dentist should train his patient in the method of using the brush. The gingival borders should not only be stimulated, but the bristles should be passed in between the gum margin and the tooth so as to remove the debris and exfoliated epithelial scales which have accumulated therein. These are often the cause of the irritation. Unless this is done the gum or epithelial tissue cannot perform its functions or be restored to health. Stimulating astringents and germicidal mouth washes should be employed whenever the gums are massaged. One of the best gum washes is that suggested by Dr. W. H. Whitslar,[5] of Cleveland, Ohio, the principal drug of which is sulphocarbolate. This drug may be used in different strengths and in many forms. I use the following:

Gum Wash.

Zinc sulphocarbolate	gr. 60
Alcohol	oz. 1
Distilled water	oz. 2
True oil of wintergreen	gtts. 8

The massage should be done three times a day. The patient should be under the care of the dentist at least twice or thrice a week, so that he may direct the treatment. If the teeth are irregular, care and patience are required to reach the festoons between the teeth. After the gums are in perfect health, the patient should visit his dentist at least four times a year, or even oftener if necessary, for inspection. If on inspection the gums be found diseased at any point, the dentist can direct the attention of the patient to the particular locality and the disease be eradicated. By this method and this alone can the gums be

[5] Dental Summary, 1907, No. 8.

kept in a healthy condition. Each patient must be given specific directions as to the treatment of his or her case.

When the patient seeks our services, we should decide by a thorough examination whether the disease is due to local or constitutional causes or both. The age of the patient, condition and character of deposits, if any, condition of mouth, jaws and dental arches, condition of urine, occupation and everything pertaining to the patient should be considered. While this examination is being conducted, which requires a few days, the local treatment may be undertaken. This consists of an application to the mouth, gums, mucous membrane and teeth of a germ destroyer every other day until the parts are in an aseptic condition as far as possible.

I cannot here too strongly condemn the method of starting the treatment of this disease by scraping the roots of teeth which may or may not have calcic deposits, wounding the soft tissues and infecting them without first rendering them as aseptic as possible. Asepsis is always the first precept of the surgeon before any kind of an operation. Why should the dentist be exempt from similar methods? It is fortunate for the dentist that stasis of blood prevents infection to a great extent or serious results might occur.

This, however, dees not justify the vicious treatment employed, since the method of procedure is wholly unscientific and not in harmony with good practice.

THE IODIN TREATMENT.

The operator is not justified in placing his fingers in the filthy mouths of his patients. This is also true of the students in the clinics of our dental schools. It is in the clinic where the student should be taught the object lesson of cleanly mouths before operations. No patient in the clinic or in private practice should be operated upon before a thoroughly aseptic mouth has been obtained. The method is so simple and so easily performed that only a few minutes is required before operation may be commenced. This treatment should consist of the free use of iodin applied to the gums and teeth, carrying it well up into the pockets. This will destroy every germ with which it comes in contact. After many years of experimentation, sur-

geons have come to realize that iodin is the quickest acting and best germicide we now possess. The author, when he began his researches in 1878, commenced the use of iodin and has used it to the exclusion of all other drugs in the treatment of this disease. The results obtained are all that can be desired.

The official tincture of iodin contains seven per cent of iodin dissolved in alcohol to which is added five per cent of potassium iodid. This preparation, if used often, will cause the membrane to become tender and sore; it will also, in some patients, destroy the mucous surface. To overcome this difficulty, many years ago, I formulated the following which I have called iodoglycerole:

Zinc iodid	15 parts or grams
Water	10 parts or grams
Iodin	25 parts or grams
Glycerin	50 parts or grams

As compared with the ordinary tincture of iodin, its astringent and antiseptic properties are greatly increased, the glycerin causes rapid absorption and the irritating effects are reduced to a minimum. The penetrating effect is remarkable. The glycerin thickens the preparation and prevents it from mixing with the saliva and running over the mouth as the ordinary tincture will do. Long, round, wood applicators can be obtained at the drug and instrument houses and on one end cotton is wound; this is saturated with the preparation and the gum margins above and below painted. The jaws are closed, the lips and cheeks distended and the application made as before; the teeth are also covered; the lips and cheeks are held away from the jaws until the iodin has dried.

These applications should be made every other day and continued until the patient is dismissed. In a fairly clean mouth, the process of removing the local causes may be commenced at the second appointment or possibly at the first sitting after the iodoglycerole has become dry. In the more filthy mouths, the time to commence operations will depend upon the judgment of the operator and the condition of the mouth under treatment.

In those patients who are having operations upon the teeth,

although their gums are in fairly good condition, they are treated after each sitting to destroy lactic-acid bacilli and their ferment and all other foreign and undesirable material in the mouth, thus preventing tooth decay. By this method of procedure, I have reduced decay of the teeth from thirty to forty per cent in my patients in the past ten years. While this treatment is being conducted by the operator, the patient should use the gum wash twice a day as directed. After the patient has been dismissed the gum wash should be used continually once a day.

Having destroyed all the germs in the mouth including pus germs (except perhaps in so-called pockets out of reach with the iodoglycerole) and contracted the gums, more or less, about the necks of the teeth and on to the bone, exploration of the mouth for local irritants and irregularities may now be undertaken.

Erupting Teeth, whether the first or second set, should be examined by the operator and the inflamed gums receive the iodoglycerole treatment. This procedure should be continued in the public schools among the poor children as well as at the homes of the well-to-do. If the iodoglycerole gum bath was introduced into the public schools, contagions and infections would be reduced to a minimum.

MODERN DENTISTRY.

I have stated that modern dentistry has produced disease of the gums, pericemental membrane, and alveolar process more than any one cause. The education of the student in the dental schools in the mechanics of dentistry has been conducted to the exclusion of the pathology of the mouth. The mechanics have been carried far beyond normal physical tolerance. The discovery that pus formation in alveolar, pericemental and blind abscesses and the accumulation of pus about the alveolar process and roots of the teeth cause many of the diseases of the human body is a just evidence of the necessity of a medical education of the dentist. Not until the methods of teaching have been reconstructed, can we expect any improvement from the pathologic standpoint in the management of the mouth and teeth. The local causes which bring about interstitial gingivitis have been discussed in Chapter XVII and every teacher and practitioner is familiar with them, hence it is hardly necessary to

discuss the question here. It is to be hoped that the profession will soon consider the pathology of our specialty and so far as possible correct and prevent local irritations, inflammations and abscesses.

In passing this subject, however, there are a few suggestions which may be offered here. There is no excuse for the wholesale movement of teeth without extraction. The specialist should make a study of the jaws and teeth of each patient to ascertain how much may be accomplished with as little movement of the teeth as possible. This is to be frequently accomplished by the sacrifice of one or more teeth. The health of the patient, as well as the causing of as little inflammation in the alveolar process as possible, should be the foremost thought in the mind of the operator. The frequent use of iodoglycerole is indicated during the operation of correcting irregular teeth to keep down the inflammation. After the retaining bands have been adjusted the iodoglycerole should often be used.

We have shown that the condition of the alveolar process, especially after it has attained its growth and its endo-transitory nature, is a very unfavorable structure for the successful operation for the implantation or transplantation of teeth, particularly so when the peridental membrane is not present.

Gold Crowns and Bands should only extend to the gum margin and never above it except in extreme cases.

Artificial Dentures should be so constructed as to produce the least amount of irritation possible. The larger the surface the better the adaptation. Iodoglycerole treatment to the surface of the mucous membrane under the plate to destroy germs and reduce inflammation should be frequently applied.

Individual Teeth whose function is not restored from want of proper articulation—too great or not sufficient pressure—must be corrected. Such teeth are liable to become diseased, like any other organ or structure of the body when not properly exercised takes on pathologic changes according to the tissue involved.

Tartar upon the teeth acts as a local irritant and should always be removed. In some mouths it accumulates rapidly and must be removed as often as once a month; in other mouths no

tartar is present. The teeth in such patients should be cleaned as often as twice a year. These suggestions, however, are only given out approximately, since the operator must decide by the conditions as they exist how often visits should be made to obtain the best results. Having destroyed the germs in the mouth and about the teeth by the free use of iodoglycerole, the tartar may be removed without fear of infecting the tissues and with a large degree of comfort to the operator on account of a clean mouth. Laceration of the gums in performing this operation is desirable. It removes the excess of stagnant blood from the tissues and greatly assists in restoring normal circulation. Three decades ago, I advocated the following set of scalers:

They consist of handles, shanks bent at different angles, and three cornered blades, so that they can be used in three directions without removing the fingers from the tooth (Fig. 100).

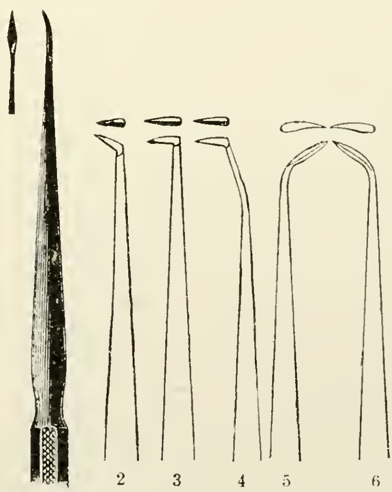


FIG. 100.—THE AUTHOR'S SET OF SCALERS.

These are all delicately made and tempered very hard. Sharp edges are thus retained. They will reach every point where tartar can collect. After the deposits have been fairly well removed, the gums may be syringed with hot water to remove the debris. The gums should then be saturated with iodoglycerole and the patient dismissed. Applications should be made every two or three days. The gums will contract and healthy circulation follow. The gum massage with the gum wash will

now be used twice a day. After a few treatments and the constant use of the massage brush, the gums will contract and other deposits which were not perceptible at the first sitting will now be presented to view. This method of treatment should be continued until the gums and alveolar process are restored to health. The patient should return to the dentist as often as necessary (every month or two) to have the gums examined and for further instructions.

As has been already shown, chronic interstitial gingivitis may extend only to the peridental membrane, to the periosteum, or it may extend throughout the alveolar process with the absorption of the bone the entire length of the root of the tooth. Pus infection and calcic deposit may or may not take place. In the early stages of this progressive inflammation, the first is probable. If pus and deposits are present they can be treated with signal success according to the symptoms and as hereinafter explained. As already suggested, the gum massage brush must be vigorously used to relieve the engorged tissues of blood. Since absorption of the alveolar process depends upon irritation and inflammation, this must be removed as quickly as possible.

In the treatment of deep-seated interstitial inflammation, iodin or iodin and aconite has always been regarded by physicians and surgeons as the best remedy. The gums should be thoroughly saturated twice or thrice weekly, as already suggested. If the alveolar process be so absorbed that the tooth has become loose, the case is hopeless. In such unstable tissues, especially when inflammation extends through the process and lacunar, perforating canal absorption and halisteresis is going on, reversal of the order so as to set the osteoblasts to tissue-building is hardly to be expected. The tendency is to destroy and not restore the alveolar process. In such cases the tooth must be fastened to the other teeth perfectly tight to prevent motion in any direction. The movement of the tooth in mastication intensifies the irritation, which in time only increases the absorption. Liberal use of iodin and the gum massage brush is all that can be done to reduce the inflammation and absorption as much as possible. The exfoliation is only a matter of time. If the tooth or teeth cannot be retained perfectly tight, no mat-

ter how healthy the surrounding tissues may be restored, the irritation produced by the loose teeth will soon set up inflammation in the surrounding tissues. The sooner the loose teeth are removed the better. In no case can the bone tissue be restored, if the matrix or cartilage be destroyed, since in this the osteoblasts are located. If the matrix or cartilage be destroyed, a fibrous union (such as occurs in the case of implanted teeth and the imbedding of foreign bodies in the tissues of the body) only is possible.

If inflammation has extended into the periosteum, peridental membrane and alveolar process, calcic deposits are frequently found upon the roots of the teeth. When this has taken place, the calculus must be removed. This should be done with the utmost care, in order that adjacent tissues may not be injured, or inflamed parts infected with pus germs. Since dead bone is not present, the operator should confine his instrumentation entirely to the root or roots of the teeth, with as little injury as possible to the adjacent tissues. The alveolar process must

Fig. 101.—Spoon Shaped Excavators for Scaling the Roots of Teeth.

under no consideration be touched. Riggs believed that the edge of the alveolar process was always in a state of disintegration, and that it should be so scraped as to get a fresh surface, on the principle of caries of bone. Many dentists are operating in this manner at the present time. I have elsewhere shown simple absorption and not caries is present. Such treatment is wholly unnecessary and contraindicated. The object of the removal of the deposits is to allow the fibrous tissue of the peridental membrane (after health is restored) to tighten about the root, which cannot be accomplished when foreign substances are present.

Pushing instruments must never be used, but only such instruments as have smooth and round backs, tempered very hard so as to retain sharp edges. These instruments should be small, with small points to reach depressions, and to be as universal as possible. Such an instrument is to be found in the spoon excavator (Fig. 101). The shank can be bent to suit the operator. This is to be carried gently along the length of the root and passed over the deposit with a firm hand, resting the finger upon some other teeth. The drawing motion is invariably to be from the membrane, and toward the crown. Attention was first called to the mutilation and infection of tissues two decades ago, at which time most, if not all, instruments for the removal of deep-seated calcic deposits were used with the pushing movement.[6] The deposits are scaled off painlessly. The round blade being larger than the shank, and cutting upon three edges, half of the root in both directions can be circled without removing the instrument. A similar instrument bent at the shank in the opposite direction may be used on the other side. After all of the roots of the teeth have been scaled, the spaces are to be syringed out with warm or hot water. The gums are to be thoroughly saturated inside and out with iodin. The gum massage brush is to be used thrice daily as before. The patient should return twice or thrice a week for further instructions. The contracting gums will assist greatly in revealing the deposit. If deposits still remain on the roots (the appearance of the gums will indicate its presence) further use of the scalers is indicated. The delicate instruments and the accustomed sense of touch will reveal the hidden calculus.

With the precautions already noted, local anæsthesia is unnecessary. The smooth, round surface of the back of the instrument, if carefully inserted, will not produce pain.

If the gum be painful to the touch, or if the patient be nervous and sensitive, application of iodin may be used, together with massage, for a few days before scaling is resorted to. The sensitiveness will soon disappear, when the instrument may be inserted without difficulty.

[6] Jour. Am. Med. Assoc., Jan. 16, 1897.
[7] International Dental Journal, April, 1896.

In an article upon "Pyorrhœa Alveolaris,"[7] I showed the difficulty of removing the deposits upon the roots of the teeth with instruments and made the following statement: "From our past experience in the treatment of the disease, the deposits must be removed; and right here I would suggest that in the future treatment of this disease a dissolving fluid that is not injurious to the surrounding tissue should take the place of instruments, especially when the disease is extensive."

I am pleased to state at this time that Dr. Joseph Head, in a paper, "A Tartar Solvent, Especially Useful in Pyorrhœa Work,"[8] in which he demonstrated his experiments upon the action of acid ammonium fluorid as a solvent for calcic deposits, claimed to produce good results without action upon the tooth structure or soft tissue. This preparation, known as "Tartasol," can be obtained at any of the dental depots. This or similar drugs and the method of application must eventually become the proper treatment for the satisfactory removal of deposits upon the roots of the teeth. In this way and this alone can we expect to obtain a clean, smooth root surface. A few years hence, the profession will regard a dentist whose patients have pus oozing from the gums as a prehistoric relic and the patient as an individual whose filth provokes the contempt of his fellows. In this day of antisepsis, the dentist is as accountable for pus infection of his patients as the physician or surgeon.

There is no more excuse for the dentist's patient being infected than the surgeon's. If ordinary antiseptic precautions are taken, pus infection will not often occur. Prevent inflammation of the gum margin and pus infection cannot follow, no matter how many germs are in the mouth. This is an absolute law of general pathology. It has been proven by the experiments made by Miller, G. T. Carpenter and myself on dogs, rabbits, guinea pigs and man.

The illustrations of the progress of interstitial gingivitis teach that only the mildest treatment is indicated. Harsh treatment on the inflamed bone or fibrous tissue, either with instruments or drugs, must not be employed. Heroic treatment, such as the indiscriminate application of sulphuric and lactic acid and

---

[8] Transactions National Dental Association, 1899, p. 131.

similar drugs in nearly or quite full strength, is not justified by the surgical principles of today. No surgeon would think of making such an application to inflamed bone in other parts of the body unless he wished necrosis with a desired sequestrum. Much less would the intelligent operator use such treatment in a transitory structure which predisposed to destruction. In a number of instances exfoliation of the anterior plate of the alveolar process has resulted from this treatment, to say nothing of the intense pain produced. J. M. Whitney[9] has had four cases in his practice in which serious results followed. The first indication is to remove the cause. Instrumentation should be resorted to only to remove tartar and calcic deposits. This must be done in such a manner as not to infect the deeper inflamed tissue or carry the products of inflammation into healthy tissue. The treatment of infected tissue within and about the alveoli is not unlike treatment of abscesses and ulceration elsewhere. Such drugs as are used in abscesses and ulcers in other tissues are indicated here in the same strength. If strong drugs be used they should not be permitted to remain in the tissue, lest necrosis of the alveolar process occur. They must be diluted or removed altogether after they have accomplished their purpose Very serious results have occurred from careless use of drugs. When abscesses have formed they should be opened and hydrogen peroxid—or, which has answered my purpose equally well, hot water—is all that is necessary. More difficult is treatment of ulceration of the tissue near the root of the tooth. Ordinary cases will heal after hot water or hydrogen peroxid have been applied. In some cases the pus germs have followed the inflammation along the course of the vessels quite a distance into the interstitial tissue. In such cases they are difficult to reach. A small syringe may be employed, or the drug may be carried to the part on the end of a long, thin orange-wood stick. In all cases the drug must be directly applied to the part in order to have beneficial results. Applications of iodin should be used, as already suggested. Iodin carried to the ulcerated surface often suffices to destroy the pus secretion. Ordinarily one or two applications is sufficient. Occasionally calcic deposits are

[9] International Dental Journal, April, 1899.

located in front of the infected surface and the drug does not reach the part. In such cases the deposit must be removed. If the pus does not cease at the first, second or even third application, this is not because the drug is not sufficiently strong, but because it does not reach the infected part. Continued applications of iodin externally and internally, carried well up between the roots of the tooth and the alveolar process will, in time, produce the desired result. When pus ceases to flow, antiseptic treatment must stop. The iodin and massage treatment must then be pushed until the interstitial inflammation has been reduced and the gums contracted tightly about the necks of the teeth.

After the tissues have been placed in a healthy condition, they will require the constant attention of the operator, since, like other tissues of the body when once diseased, favorable conditions will cause a recurrence. The patient must return to the operator frequently so that he can advise as to the use of massage.

When constitutional disorders are the cause of interstitial gingivitis, local treatment will not cure the disease. It is possible to deplete the parts of blood and reduce the inflammation to a minimum. The cause not having been removed, the inflammation soon returns.

When the disease is due to constitutional causes, tartar deposits rarely occur; scraping the roots, therefore, is useless. Pus germs may or may not be present. The local iodoglycerole treatment, however, is indicated, but the constitutional causes of the disease must be considered by a competent physician. The history of the patient must be looked into, a complete urinary examination made and the heart pressure taken. The heart, liver, kidney, bowels, lungs and skin must be placed in a healthy condition; without this attention local results are impossible. Change in climate and food frequently benefit the patient. Loose teeth must be fastened tightly to other teeth; this, however, is only temporary, since (as I have already mentioned) the function is lost. Better results can be obtained by their removal.

VACCINE TREATMENT OF INTERSTITIAL GINGIVITIS.

In the treatment of a disease by vaccine, it is positively necessary that the exact nature and identity of the germ or germs producing the disease be known. It is also necessary that the various types of infections as well as the pathologic condition in which the bacteria are present as secondary infections be known.

This knowledge will prevent the specialist from using a vaccine which will not immune the germs which produce the disease.

The technique of this treatment is of so much importance that I cannot express my views better than to quote from an article, "The Principles of Bacterin Therapy," by Dr. J. Favil Biehn,[10] "The corresponding bacterins are indicated in all bacterial infectious diseases; but since the bacterins have a specific action only, it is absolutely essential to know the particular organism or organisms causing the disease under treatment and to give the corresponding bacterins. Thus, for instance, bacillus-coli bacterins are of value only in diseases caused by the bacillus coli; they are practically valueless in diseases caused by other organisms. Hence, there are no bacterins for such ailments as boils, furuncles, and the like, for these conditions are not always produced by the same organism or group of organisms. In one case streptococci, in another case staphylococci (either albi or aurei) may be the etiological cause. The first form will be benefited only by the bacterins containing streptococci, while the second responds only to one containing staphylococcus albus, etc. However, a mixed bacterin may be employed, and will prove beneficial, provided it contains the specific organisms responsible for this particular diseased condition.

"Therefore, unless the exact etiologic cause is determined and the corresponding bacterins are administered, failure will surely result. It is quite necessary, in case several pathogenic bacteria are acting together in producing a disease, that the corresponding bacterins for all of them be utilized; and further, if the infection should change as a result of the invasion of other organisms unless a bacterin for these organisms is also used, a complete cure may not be obtained."

---

[10] American Journal of Clinical Medicine, February, page 157.

A careful study of the researches in this work will show that interstitial gingivitis is due to local and constitutional irritants and toxins and not to infections.

The operator must not lose sight of the fact that it is the inflammatory condition which causes the absorption of the alveolar process, and the exfoliation of the teeth and not the pus stage. The pus formation is the result and not the cause of the disease.

While it is possible that a vaccine may be made, of the pus germs which cause the secondary state of the disease (pyorrhœa alveolaris), which may possibly render these germs innocuous and stop the flow of pus, such treatment can, according to our present knowledge, hardly be expected to reduce the primitive stage (interstitial gingivitis) so much to be deplored. It is barely possible that in the future specific germs may be discovered which may cause the inflammatory stage. When this has been accomplished, a vaccine may be produced which will be a positive method of treatment. Until then the vaccine method of treatment should be used with discretion.

BIBLIOGRAPHY

The following books and monographs of the author have been drawn on largely for material in compiling the present work.

BOOKS

1. The Irregularities of the Teeth, First Edition, 1888.
2. The Irregularities of the Teeth, Second Edition, 1890.
3. Chart of Typical Forms of Irregularities of the Teeth, 1891.
4. A Study of the Degeneracy of the Jaws of the Human Race, 1892.
5. The Etiology of Osseous Deformities of the Head, Face, Jaws, and Teeth, Third Edition, 1894.
6. Degeneracy: Its Signs, Causes and Results (London), 1898.
7. Interstitial Gingivitis or So-called Pyorrhoea Alveolaris, 1899.
8. Irregularities of the Teeth, Fourth Edition, 1901.
9. Quiz Compend of Irregularities of the Teeth, 1901.
10. Irregularities of the Teeth, Fifth Edition, 1903.
11. Developmental Pathology; A Study in Degenerative Evolution, 1912.

MONOGRAPHS

1. Education, Dental Colleges — *The Dental Cosmos,* 1876.
2. Mercury, Chemical and Physiological Action of Fillings on the System—*The Dental Cosmos,* 1879.
3. Preparation of Nerve Canals for Treatment and Fillings — *The Dental Cosmos,* 1880.
4. Gold Crowns—*The Dental Cosmos,* 1880.
5. Screws for Artificial Crowns—*The Dental Cosmos,* 1881.
6. Treatment and Filling of Approximal Cavities—*The Dental Cosmos,* 1881.
7. The Regulation of Teeth by Direct Pressure — *The Dental Cosmos,* 1881.
8. Dental Regulating Apparatus—*The Dental Cosmos,* 1885.
9. Spreading the Dental Arch—*The Dental Cosmos,* 1886.
10. Regulating Individual Teeth — *The Dental Cosmos,* 1886.
11. Pyorrhœa Alveolaris, 1st Paper—*The Dental Cosmos,* 1886.
12. The Etiology of Irregularities of the Teeth—*The Dental Cosmos,* 1888.
13. Arrest of Development of the Maxillary Bones, due to Race Crossing, Climate, Soil and Food — *The Dental Cosmos,* 1888.
14. Development of the Inferior Maxilla by Exercise and Asymmetry of the Lateral Halves of the Maxillary Bones — *The Dental Cosmos,* 1888.
15. Asymmetry of the Maxillary Bones —*The Dental Cosmos,* 1888.
16. The Alveolar Process—*The Dental Cosmos,* 1888.
17. The Origin and Development of the V and Saddle Arches and Kindred Irregularities of the Teeth—*The Dental Cosmos,* 1889.
18. The Above Concluded—*The Dental Cosmos,* 1889.
19. Classification of Typical Irregularities of the Maxillæ and Teeth—*The Dental Cosmos,* 1889.
20. Statistics of Constitutional and Developmental Irregularities of the Jaws and Teeth of Normal, Idiotic, Deaf and Dumb, Blind and Insane Persons—*The Dental Cosmos,* 1889.
21. Fallacies of Some of the Old Theories of Irregularities of the Teeth with Some Remarks of Diagnosis and Treatment—*The Dental Cosmos,* 1890.
22. The Teeth and Jaws of a Party of Cave and Cliff Dwellers—*The Dental Cosmos,* 1890.
23. The Differentiation of Anterior Protrusions of the Upper Maxilla and Teeth, International Medical Congress, Berlin — *The Dental Cosmos,* 1890.
24. Mouth-Breathing not the Cause of Contracted Jaws and High Vaults —*The Dental Cosmos,* 1891.
25. Management of Dental Societies—*The Dental Cosmos,* 1891.
26. Studies of Criminals—*The Alienist and Neurologist,* 1891.
27. Scientific Investigation of the Cranium and Jaws—*The Dental Cosmos,* 1891.
28. Evidence of Somatic Origin of Inebriety—*Journal of Inebriety,* 1891.

29. A Study of the Degeneracy of the Jaws of the Human Race—*The Dental Cosmos*, 1892.

30. Empyema of the Antrum—*Journal of the American Medical Association*, 1893.

31. The Vault in its Relation to the Jaw and Nose—*The Dental Practitioner and Advertiser*, 1894.

32. Stigmata of Degeneracy in the Aristocracy and Regicides—*Journal of the American Medical Association*, 1894.

33. The Degenerate Ear—*Journal of the American Medical Association*, 1895.

34. Pyorrhoea Alveolaris, 2nd Paper—*International Dental Journal*, 1896; *The Dental Cosmos*, 1896.

35. Dental and Facial Evidence of Constitutional Defect—*The International Dental Journal*, 1896.

36. H. H. Holmes—*Journal of the American Medical Association*, 1896.

37. Pyorrhoea Alveolaris, 3rd Paper—*Journal of the American Medical Association*, 1896.

38. Degeneracy of the Teeth and Jaws—*Journal of the American Medical Association*, 1896.

39. Oral Hygiene—*International Medical Congress, Moscow*, 1897.

40. Autointoxication in its Medical and Surgical Relations to the Jaws and Teeth—*Journal of the American Medical Association*, 1897.

41. Pyorrhoea Alveolaris in Mercurial and Lead Poisoning and Scurvy, 4th Paper—*Journal of the American Medical Association*, 1898.

42. Degeneracy in its Relation to Deformities of the Jaws and Irregularities of the Teeth—*The Chicago Dental Review*, 1898.

43. A Study of the Stigmata of Degeneracy among the American Criminal Youth—*Journal of the American Medical Association*, 1898.

44. Irregularities of the Dental Arch—1898.

45. A Study of the Deformities of the Jaws among the Degenerate Classes of Europe—*The International Dental Journal*, 1898.

46. Inheritance of Circumcision Effects—*Medicine*, 1898.

47. What Became of the Dauphin Louis XVII? A Study in Dental Jurisprudence—*Medicine*, 1899.

48. Interstitial Gingivitis due to Autointoxication — *The International Dental Journal*, 1900.

49. Traitement de la Pyorrhie Alveodentaire—*XIII International Medical Congress Proceedings*, Paris, 1900.

50. The Intervention of Therapeusis in Anomalies of Position and Direction of the Teeth—*XIII International Medical Congress Proceedings*, Paris, 1900.

51. Limitations in Dental Education—*Journal of the American Medical Association*, 1900.

52. Interstitial Gingivitis from Indigestion Autointoxication—*Journal of the American Medical Association*, 1900.

53. Interstitial Gingivitis as a Prominent Obvious Early Sympton of Autointoxication and Drug Poisoning—*Chicago Medical Society Proceedings*, 1901.

54. Peridental Abscess — *Proceedings New York State Dental Society*, 1901. *The Chicago Dental Review*, 1901.

55. Oral Manifestations and Allied States—*Journal of the American Medical Association*, 1901.

56. Degeneracy and Political Assassination—*Medicine*, 1901.

57. The Higher Plane of Dentistry—*Revue de Stomatologie*, Paris 1902.

58. Juvenile Female Delinquents—*The Alienist and Neurologist*, 1901-2-3.

59. The Stigmata of Degeneracy—*The Medical Examiner and Practitioner*, 1902.

60. Deformities of the Bones of the Nose and Face—*The Laryngoscope*, 1902.

61. Evolution of the Pulp—*Journal of the American Medical Association*, 1902.

62. Why Dentists do not Read—*The International Dental Journal*, 1903.

63. How Far do Stomatologic Indications Warrant Constitutional Treatment? — *The International Dental Journal*, 1903.

64. Syphilitic Interstitial Gingivitis—*The International Dental Journal* 1903.

65. Gum Massage—*The International Dental Journal*, 1903.

66. The Vasomotor System of the Pulp—*Journal of the American Medical Association*, 1903.

67. Recognition of the D. D. S. Degree by the American Medical Association—*Dental Journals*, 1903.

68. What the Physician or Surgeon should know of Dentistry—*Illinois Medical Bulletin*, 1903.

69. Pathogeny of Osteomalacia or Senile Atrophy—*Journal of the American Medical Association,* 1904.
70. Constitutional Causes of Tooth Decay—*The Dental Digest,* 1903.
71. Interstitial Gingivitis or So-called Pyorrhoea Alveolaris—*The Dental Summary,* 1903.
72. Buccal Expressions of Constitutional States—*Medicine,* 1903. *The Dental Digest,* 1903.
73. Endarteritis Obliterans and Arterio-Sclerosis of the Alveolar Process —*The Dental Digest,* 1903.
74. Pathology of Root Absorption and Alveolar Abscess—*The Dental Digest,* 1904.
75. The Relations of the Nose and Genitalia—*Medicine,* 1904.
76. Pulp Degeneration—*Journal of the American Medical Association,* 1904.
77. Criminal Responsibility and Degeneracy—*British Medical Association, Section on Psychological Medicine,* 1904.
78. Anatomic Changes in the Head, Face, Jaws and Teeth in the Evolution of Man—*Fourth International Dental Congress,* St. Louis, Mo., 1904.
79. Constitutional Causes of Tooth Decay, Erosion, Abrasion and Discoloration—*Fourth International Dental Congress,* St. Louis, Mo., 1904.
80. Etiology of Cleft Palate—*Fourth International Dental Congress,* St. Louis, Mo., 1904.
81. Scorbutus or Interstitial Gingivitis —*Medical News,* 1904.
82. Negro Ethnology and Sociology—*Illinois Medical Bulletin,* 1905.
83. Gonorrhoeal Ulcero-Membraneous Stomatitis — *The International Dental Journal,* 1905.
84. Evolution of the Central Nervous System—*The Dental Digest,* 1905.
85. Study of the Pithecanthropus Erectus or Ape-Man—*The International Dental Journal,* 1905.
86. Advance and Retrogressive Evolution—*The Dental Digest,* 1905.
87. Underlying Factors of Developmental Pathology or Suppressive Evolution—*The Dental Digest,* 1905.
88. Laws Governing Eugenesis: A Thirty-five Years Study of Developmental Pathology—*The Dental Era,* 1905.
89. Developmental Pathology and Tooth Decay—*The Dental Cosmos,* 1905.
90. Errors in Dental Education—*The Dental Cosmos,* 1906.
91. Interstitial Gingivitis due to Autointoxication: Etiology—*The Dental Digest,* 1906.
92. Interstitial Gingivitis due to Autointoxication as Indicated by the Urine and Blood Pressure Diagnosis—*The Dental Digest,* 1906.
93. Therapeusis and Treatment of Interstitial Gingivitis due to Autointoxication—*The Dental Digest,* 1906.
94. Acid Autointoxication and Systemic Disease the Cause of Erosion and Abrasion—*Proceedings of the New York State Dental Society,* 1907.
95. Alcohol in its Relation to Degeneracy—*Journal of the American Medical Association,* 1907.
96. Acid Intoxication or Acidosis: A Factor in Disease—*New York Medical Record,* 1907.
97. Stomatology in its Medical Aspects —*Extrait du Bulletin of the International Association of Stomatology,* Bruges, 1908.
98. Swan Songs and Degeneration of American Dental Colleges—*The Dental Cosmos,* 1908.
99. The Care of the Teeth—*Illinois Medical Bulletin,* 1908.
100. Etiology of Face, Nose, Jaw and Tooth Deformities—*Journal of the American Medical Association,* 1909.
101. Bone Pathology and Tooth Movement—*Journal of the American Medical Association,* 1909.
102. Acidosis, Indicanuria, Internal and External Secretions: the Effects upon the Alveolar Process and Teeth—*The Dental Cosmos,* 1908.
103. Sense and Nonsense as taught in American Dental Schools—*The Dental Cosmos,* 1909.
104. Treatment of Interstitial Gingivitis —*The Dental Cosmos,* 1909.
105. Progress of Stomatology in Europe—*The Dental Cosmos,* 1909.
106. Hard Teeth and Soft Teeth—*The Dental Cosmos,* 1909.
107. Progress of Stomatology in Hungary —*American Journal of Clinical Medicine,* 1909.
108. Local Manifestations of Systemic Diseases—*XVI International Medical Congress, Budapest,* 1909.
109. How Shall the Stomatologist be Educated?—*International Association of Stomatology,* Budapest, 1909.
110. Scope of Developmental Pathology —*The Alienist and Neurologist, Feb.* 1910.
111. Rip Van Winkles in American Dental College Faculties—*The Dental Cosmos,* 1910.

112. The Scope of Developmental Pathology in its Relation to Oral Manifestations—*International American Congress of Medicine and Hygiene,* 1910.

113. Oral Hygiene in American Dental Schools — *The Dental Cosmos,* 1910.

114. The Acidemic Condition—*The Medical Standard,* 1910.

115. Iodin as an Astringent, Antiseptic, Disinfectant and Germicide in the Treatment of Mouth Diseases—*Journal American Medical Association,* 1910.

116. How Shall the Stomatologist be Educated?—*Journal American Medical Association,* 1910.

117. Care of the Mouths of School Children—*The Dental Summary,* 1910.

118. The Quality of Service Rendered—*The Dental Summary,* 1910.

119. What are Dentists as a Profession doing to Advance their Specialty? —*The Dental Summary,* 1910.

120. Treatment to Alleviate the Contagions, Infections and Local Diseases of School Children—*The Dietetic and Hygienic Gazette,* 1910.

121. Developmental Pathology: A Study in Degenerative Evolution—*Proceedings of the First District Dental Society of New York,* 1910.

122. The Future of Dentistry—*Sunday Editorial in The Chicago Tribune.* 1911.

123. Some Bacterial and Non-Bacterial Diseases—*Dental Summary, June* 1912.

124. The Relation of Rheumatic Arthritis to Pyorrhoea Alveolaris—*Clinical Medicine, November,* 1912.

125. Degeneracies, the Result of Alcohol and other Narcotics—*Read before Society for the Study of Alcohol and Other Narcotics, at Washington, D. C., Dec.* 11-12, 1912.

126. Interstitial Gingivitis and Pyorrhoea Alveolaris — *Journal American Medical Association,* 1913.

INDEX OF AUTHORS

INDEX OF SUBJECTS